THE MASTERWORK OF JULES TISSOT

ENGLISH EDITION

VOLUME II

This translation of J. Tissot's 1936 work 'Constitution Des Organismes'
is based on the original French edition, which is in the public domain.
Translated by **Someone for Everyone** in 2025.
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Dedication

To all who search for meaning
in the smallest of things, and to
the unseen forces that give life
its Beginning and its End

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''The oldest contagious disease known to affect humans is tuberculosis, a respiratory disease caused by *Mycobacterium tuberculosis*.

The bacterium is thought to have existed in something similar to its modern form for more than 70,000 years, and has been infecting humans since early prehistory.

In 2007, a group of scientists published a paper that reported signs of tuberculosis infection in a 500,000-year-old *homo erectus* skeleton.

Egyptian mummies from 2400 BC show signs of tuberculosis, and written evidence from India that describes tuberculosis, survives from 3300 years ago.

In ancient Greece, tuberculosis was known as Phtisis and was described by Greek physician Hippocrates as particularly fatal to young adults.

The Greek physician Clarissimus Galen, who treated the Roman Emperor Marcus Aurelius in 174 AD, wrote about tuberculosis and recommended fresh air, milk, and sea voyages as effective treatments. ''

Guinness World Records
- the oldest contagious disease.

"Education is the most important thing," he told me once.
"Not just for me, you know, but also for the nation."

John Green (Everything Is Tuberculosis:
The History and Persistence of Our Deadliest Infection)

"While tuberculin cannot treat TB,
it can identify TB, because only those who are infected
with M. tuberculosis will have an immune response to tuberculin."

John Green (Everything Is Tuberculosis:
The History and Persistence of Our Deadliest Infection)

''Today, more than ever before, the authoritarian world is waiting for salvation from the test tube. But even if a vaccine were to be found, there is another problem: as AIDS most probably originated through genetic engineering, only an antidote on the same basis will inevitably be able to offer protection and supposed help. The scenario when this new vaccine comes onto the market is foreseeable: practically the entire world population will be genetically engineered in the largest human trial in history, with an uncertain outcome. No one will be able to predict the pitfalls and long-term effects of this messiah of free love''

Wolfgang Eggert ''The Planned Plagues'' (2003)
(Translated from German.)

J. TISSOT
Professeur de Physiologie Générale
au Muséum national d'Histoire Naturelle

CONSTITUTION DES ORGANISMES
ANIMAUX ET VÉGÉTAUX
CAUSES DES MALADIES
QUI LES ATTEIGNENT

•

2e VOLUME

CAUSE ET NATURE DE LA TUBERCULOSE

Origine et nature du bacille de Koch
Source originelle des virus
Constitution élémentaire des tissus

PARIS
AU LABORATOIRE DE PHYSIOLOGIE GÉNÉRALE DU MUSÉUM
D'HISTOIRE NATURELLE
7, RUE CUVIER

1936

J. TISSOT

Professor of General Physiology at the
National Museum of Natural History

CONSTITUTION OF ORGANISMS

ANIMALS AND PLANTS

CAUSES OF DISEASES

THAT AFFECT THEM

2ND VOLUME

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Origin and nature of Koch's bacillus
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7, RUE CUVIER

1936

BY THE SAME AUTHOR

Constitution of animal and plant organisms. Causes of the diseases that affect them. 1st volume.

Work comprising a volume of 679 pages and an album of 333 collotype plates.

This work includes new studies relating to:

1. The constitution and evolution of bacterial cultures.

2. The study of the transformation of bacterial cultures into mold cultures and vice versa.

3. The evolution of parasitic molds growing in the animal organism.

4. The study of the constitution of the tissues of the animal organism: connective tissue, brain, myelinated nerve fibers, muscles, liver, spleen, testicle, ovary, and the formed elements of blood.

5. The study of the constitution of plant tissues.

6. The study of the nature and constitution of ferments, toxins and antitoxins, and filtering viruses.

7. To the study of the constitution of complement or alexin.

8. To the study of the mechanism of the phenomena of hemolysis, agglutination, precipitation, and complement fixation.

9. To the determination of the mechanism of anaphylaxis.

10. To the study of the nature and mechanism of immunity against infectious diseases.

11. To the study of the causes of plant diseases, particularly diseases of potatoes, vines, cereals, and especially diseases attributed to parasitic uredinia.

12. To the study of the pathogenic agents of human diseases, and the search for the original source of these pathogenic agents.

All these studies have resulted, while correcting many erroneous findings, in providing new ones of capital importance for progress in the fight against diseases. They open a new avenue for the activity of researchers.

It is the application of the new principles established in this work that made possible the discovery of the nature and origin of tuberculosis and Koch's bacillus, which are the subject of the current second volume, and other equally important results, which will be the subject of a third volume in preparation.

This work is located in the General Physiology Laboratory of the Natural History Museum, 7 rue Cuvier, Paris.

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INTRODUCTION

The studies that are the subject of this work are the continuation of those published in 1926 under the title: "Constitution of Animal and Plant Organisms; Causes of the Diseases that Affect Them"; they constitute the second volume of this work.

I must recall that this first volume contained exclusively facts whose existence, materiality, and exact form in which they appear were recorded by photography. The main facts that were brought to light by these first studies are the following:

1. The histological constitution of the tissues of animal and plant organisms is very different from that currently accepted. I have indicated and recorded through photographs what, in reality, is the histological constitution of connective tissue, muscular tissue, the principal elements of the central nervous system, myelinated nerve fibers, the liver, the spleen, the constituent elements of blood, etc.

2. The vegetative form of these various tissues or tissue elements is in reality identical to the vegetative form of lower fungi, the hyphomycetes.

3. To complete this identity, I demonstrated, in the most formal way, after having established the development cycle which determines the reproduction and proliferation of bacteria, that bacterial cultures are transformed into hyphomycetes, that is, into mold cultures when they are placed in conditions favorable to this transformation.

I gave multiple and formal proofs of this phenomenon by carrying out the transformation into hyphomycetes of all pure bacterial cultures which are the viruses of known contagious diseases.

The reverse phenomenon also occurs. Not only can hyphomycete cultures be transformed into bacterial cultures; but, almost always, they simultaneously give rise, on the very surface of the culture medium, to very numerous bacterial elements, that is, to a bacterial culture.

4. Tissue fragments, taken under the most rigorous aseptic conditions from healthy animal and plant organisms, give rise to bacterial cultures and mycelial cultures of various forms. Here again, the demonstration was most formal and is not surprising since the vegetative form of tissue elements is the mycelial form and since a mycelial culture gives rise to a bacterial culture and vice versa.

5. These facts explain how, without the intervention of heterogeneous bacterial cultures, animal and plant bodies decompose, rot, after the death of their organization and

are transformed into bacterial and mycelial cultures which, from that moment on, become viruses capable of contaminating living animals. *This proved that all pathogenic viruses necessarily come from a living animal or plant organism that is their original source.*

6. Other very extensive research has aimed to determine the mechanism of immunity against infectious diseases and has brought to light the facts that explain what it consists of when it exists and the reason why it does not exist with regard to certain diseases.

7. A whole series of research has aimed to determine the phenomena of complement fixation, precipitation, hemolysis and the study of the bodies linked to these phenomena: alexin, hemolysins, precipitins, antibodies in general, toxins, and antitoxins.

The demonstrations I gave established, as early as 1926, the nature and physical constitution of toxins and ultra-viruses, which are constituted not by soluble products, but by very fine granulations, figurative elements formed by bacterial culture.

In this volume, it was also shown that autogenous viruses are formed in the animal organism and that a whole category of animal diseases, which were attributed to heterogeneous bacterial viruses, are, in reality, autogenous diseases accompanied by the formation of bacterial cultures by the organism itself.

The first volume presented other research relating to many other subjects; but this will not be discussed here because the results I have just presented are the only ones linked to current research.

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We know, from numerous examples, how difficult it is to have new knowledge accepted, especially when it is in opposition to erroneous facts, but accepted for more than half a century. The results that I made known in the first volume of this work were in opposition to much accepted knowledge. I knew well that many objections would be made to me, but what I did not foresee was that learned societies would judge, as was the practice several centuries ago, that my disinterested research, which clearly had only the progress of science in mind, deserved to be banned, put on the index, and this without even having been given any valid reason to justify this ban.

Learned societies have in fact judged that they would be compromising themselves by accepting the publication of the results of my research, and this at the request of official scientists.

How could I have foreseen that the facts established with as much care as I had taken for their demonstration would be judged with such severity that no one in these learned

societies was found to demand, at least, that an examination, even a superficial one, be decided before concluding that publication should be prohibited.

As a result, it was the dissemination of the new principles that I sought to introduce into science that was hindered and delayed by the action of the learned societies that I had believed were created, until then, to promote and even provoke the progress of science. Fortunately, a few free, clear, and elevated minds were found who sought to achieve this dissemination, outside of myself and without having been solicited.

These incidents, however regrettable they may be for the researcher, in the sense that they hinder his work, have not, however, prevented me from continuing to extend and develop the results acquired.

I was convinced that the new facts that I made known in 1926 in the first volume of this work, the results of which learned societies had refused to publish, were likely to lead to new discoveries and important new scientific progress in the field of general physiology , which is also that of experimental medicine.

It is therefore with great satisfaction that I come today to oppose the response of the facts to the ban imposed. This response of the facts, set out in this volume, is the discovery of the real cause of the tuberculosis, that of its spontaneous evolution which, by itself, brings down the notion of contagiousness, that of the nature and origin of Koch's

bacillus, normal mitochondria of anatomical elements, the definitive demonstration of the original source of viruses, which is the organism of animals and plants, and finally, the demonstration of the real form of mitochondria and the principal role they play in the organism.

These new acquisitions of science, made possible by the knowledge of the principles set out in the first volume of this work, constitute the striking, definitive proof of the accuracy of these principles and of the facts which establish them.

These principles do not only constitute the solution to the problems which were studied to establish them, they constitute a new path open to the scientific activity of researchers in all fields of general biology, in those of histology, general physiology and in that of all parts of medicine.

This second volume of the work "*Constitution of Animal and Plant Organisms; Causes of the Diseases that Affect Them*" must be considered as an example of the influence that the application of these principles can exert in the study of a question of general biology, when one puts at their service, in its entirety, the method of determinism which has been gradually dying out since the memorable works and teachings of **Cl. Bernard.**

One of the most important results, from a practical and immediate point of view, which I had achieved in my first research, was the determination of the original source of viruses; this source being known in the general principle, it remained to search for it for each pathogenic virus in particular and to determine exactly what was, for each of these , the animal or plant species which gave rise to it.

I had already been able to establish that *Trichophyton crateriforme* derives from an evolution of normal potato tissue and to reproduce, with certainty, this trichophytosis in rabbits by inoculation with various molds obtained by culturing the aseptic pulp of healthy potatoes.

This first success led me to believe that I would also quickly determine the original source of certain viruses, such as those of typhoid fever, diphtheria, Malta fever, etc. Taking as a starting point for research the fact that these viruses, cultivated under certain conditions, give rise to molds which can be distinguished from each other by certain characteristics linked to the specificity of their origin, I sought to obtain the molds characteristic of the main animal and plant food substances or of animal and plant species in contact with man.

Knowing that each animal or plant species can be the original source of a virus, I compared the molds obtained by transformation of known viruses with those provided by the tissues of the various animal and plant species studied. In these comparisons, I saw striking analogies that led me to believe that the Malta fever virus, for example, came from the substance of lemons or oranges, these, like cultures of *Micrococcus melittensis,* giving rise to the same species of hyphomycete, Penicilium italiaeum, easily recognizable.

By the same process, I was led to conclude that the diphtheria virus must come from barley, that of paratyphus A from wheat, that of paratyphus B from rye, that of typhoid fever from corn, etc.

I did not continue this research in the same direction because I could not provide formal proof that my conclusions were correct or convince myself of them absolutely; I judged that before continuing them in the same direction, I should turn to a virus that would provide this formal proof, vaccinia, for example. The virus obtained from its original source will provide proof, beyond any possible doubt, that it is that of vaccinia if it causes the formation of pustules analogous to the vaccinia pustule and if it achieves immunity against a subsequent inoculation with the usual heifer vaccine. I continued my research in this direction for two years without success and I am still continuing it. I thought that, if I was thus failing, it was because my knowledge of the creation of viru-

ses by their original source, and of their cultivation outside the organism that is this source, was insufficient and that it was necessary first to complete it.

Convinced that human pulmonary tuberculosis was due to a transformation in the form of the organic mold that constitutes his normal tissues, a transformation of which Koch's bacillus would be the result or the witness, I had here a case of the formation of a virus in its original source itself, if indeed I was not mistaken. I therefore chose this subject to seek to establish either the mechanism of this transformation of the normal substance of tissues into tubercular virus, which is proof of my error or of the heterogeneous origin of Koch's bacillus.

I conducted this study on the human lung and sought to establish as completely as possible the determinism of the evolution of tuberculous lesions.

During this study, I was able to arrive at knowledge of the facts which provide, by themselves, the demonstration and the clear and simple explanation:

1. Of the cause of tuberculosis and the extension of its lesions.
2. Of the cause of the destruction of lung tissue.
3. Of the origin and nature of Koch's bacillus.

These new acquisitions are of capital importance from a practical point of view because, firstly, they demonstrate that tuberculosis is a disease that arises spontaneously in humans and not by contagion. Secondly, they make it possible to clarify the role played by Koch's bacillus in the development of tuberculosis; finally, they will also make it possible, as a consequence, to definitively and safely eliminate all useless treatment methods whose aim was either to destroy Koch's bacillus, a result impossible to obtain without destroying the individual himself, or to immunize man against contagion by this bacillus, an immunization which is pointless, due to the origin of Koch's bacillus and since tuberculosis is a spontaneous, endogenous disease and not a disease contracted by contagion, at least as a general rule.

But, if these acquisitions are important from the various points of view that I have just indicated, they have an even greater importance from a more general point of view: knowledge of the mechanism of the transformation of the normal constituent elements of anatomical elements into a bacterial culture, that is, into a pathogenic virus; this fact constitutes the first demonstration of the birth of a virus in the living organism which is its original source and the first complete and direct demonstration of the fact that the original source of viruses is the substance of the organism of living beings.

The following study was carried out by pushing determinism to the solution of all the problems posed by the development of tuberculous lesions. These problems were:

1. The determination of the elements which constitute tuberculous tissue.

2. The determination of the origin, nature, and evolution of these elements.

3. The determination of the relationships that exist between the development of these elements and Koch's bacillus.

4. The determination of the nature and origin of Koch's bacillus.

5. The final determination of the real, immediate cause of natural pulmonary tuberculosis in humans.

* * *

HISTORICAL AND CRITICAL REPORT

Before **Laennec,** tuberculosis was considered the consequence of chronic lung inflammation. **Laennec** was the first to explain in 1811 that tuberculosis comes in two forms: tuberculous granulation producing a small nodule and tuberculous infiltration, which gradually invades the lung tissue without producing a nodule.

Around 1850, **Virchow (1),** following his studies of pathological anatomy, concluded that only tuberculous granulation truly relates to tuberculosis, and he considered that tuberculous infiltration, as well as *any lesion that does not have the shape of a nodule, is an inflammatory product and has no connection with the tubercle.*

Until 1865, numerous works were published, some of which supported **Laennec's** conclusions and others those of **Virchow.**

In 1865-1866, **Villemin (2)** demonstrated that inoculating a rabbit under its skin with tubercular material taken from a human, a cow, or a rabbit already tubercular reproduces tuberculosis; he thus deduced his doctrine of the contagiousness of the disease; this doctrine was considerably reinforced by the experiments of my illustrious and late master **A. Chauveau (3)** who, in 1868, succeeded in infecting three heifers by ingesting tubercular material from a consumptive cow.

Thus, was introduced into science the serious error that was to lead all future research down a fatally sterile path.

Villemin's experiments in no way included the conclusion he drew from them. This conclusion should have been strictly limited to the significance of the result of his experiments, which was as follows:

Subcutaneous inoculation of animals with tuberculous material confers tuberculosis. There was no proof in these experiments that human-to-human contagion was always the determining and necessary cause for the development of the disease under usual living conditions, which have nothing in common with the conditions of transmission by subcutaneous inoculation.

(1) *Virchow, Cellular Pathology.*
(2) *Villemin, Cause and Nature of Tuberculosis. Bull. of the Association of Med., vol. XXXII, 1866, p. 152, and Studies on Tuberculosis, 1 vol., 1868.*
(3) *A. Chauveau, Application of Knowledge of the Conditions of Infection to the Study of the Contagion of Pulmonary Phthisis, Bull. of the Association of Med., vol. XXXIII, 1868.*

As for **Chauveau's** experiments on the ingestion of tuberculous material, they only meant that infection of heifers can occur under the special conditions he created to obtain it.

Nor did they mean that humans contract pulmonary tuberculosis under living conditions that are totally different from those of his experiments.

Despite the support of **Chauveau's** experiments, **Villemin's** conclusions of regarding the contagiousness of tuberculosis were widely contested because they did not at all agree with the clinical facts observed at that time by a host of clinicians who left us proof that they were observers of the highest order.

It is interesting, from a historical point of view, to recall with what presentiment of reality **Pidoux** presented to the Academy of Medicine the arguments he opposed to **Villemin's** conclusions:

''*Experiments on animals give you this or that result, and, instead of verifying them with clinical experience and all the data of human physiology, you construct a general doctrine of human tuberculosis and all diseases on their basis. To do this, you overturn all acquired notions. We must accept, from one day to the next, that phthisis falls from the clouds and that, in its pathogenesis, the subject, the constitution, the hygienic conditions, heredity, the diatheses, are nothing, and that everything is on the blade of a lancet loaded with an impossible tuberculosis virus, undoubtedly coming from a tuberculosis patient who inherited it from another, and so on up to the first man, who nevertheless inherited it from no one and must have formed it from scratch.*''

Pidoux supported his argument by asking that a single case of tuberculosis resulting from proven contagion be made known to him. Six years later, not a single case had yet been brought to his attention.

Such arguments were of such considerable value that they left doubt, and the notion of contagiousness continued to be highly contested until 1882.

The discovery and cultivation of *Koch's bacillus* **(1)** in 1882 gave a decisive confirmation of **Villemin's** conclusions and, from that moment on, removed all objections to contagiousness; this was admitted by almost everyone.

Subsequently, the fight against tuberculosis had to be directed against the bacillus, which was necessarily believed to be the agent of tuberculosis, since it was obtained in pure culture and inoculation with this pure culture conferred tuberculosis. It must be recognized that no one would have dared to contest these conclusions and that at that time the facts seemed quite conclusive.

(1) *Koch, Die Oeliologie der Tuberkulose,, Berliner. Klin, Wocheuschr., April 10, 1882, p. 221.*

However, this discovery in no way detracted from the value of the clinical facts observed during the last century, which opposed the doctrine of contagiousness.

Since bacillary infection was almost universally accepted as the cause of tuberculosis, the fight against this disease found itself, by this fact, engaged in a path from which it could hardly escape. The study of tuberculosis was limited, from that moment on, to work on pathological anatomy with a view to clarifying particular points in the evolution of the disease.

New studies on toxins only aggravated the obscurity by leading to the admission that the evolution of tuberculous lesions was controlled by the action of the toxins of Koch's bacillus.

All attempts made, since that time, to cure tuberculosis by the most diverse methods directed against Koch's bacillus have failed; Koch's tuberculin, Behring's tests using bacilli with attenuated virulence or by injection of substances extracted from bacilli, all tests of serotherapy, have proven entirely ineffective. Only clinical practice has succeeded in making significant progress in the treatment of tuberculosis.

Currently, tuberculosis is considered by almost everyone to be due to Koch's bacillus and contagious in nature. Almost one of the few, **A. Lumière,** has, for several years, combated this notion of the contagiousness of tuberculosis in adults, by taking up the facts established by clinicians of the last century and their arguments.

I will return to this point later, which I will develop more fully.

Apart from the facts I have just presented, research in pathological anatomy is of capital importance in the study of the development of tuberculosis.

We will therefore examine specifically, here, the evolution of knowledge relating to this development.

After the work of **Virchow**, we must cite that of **Grancher (1)**, whose most important conclusion is as follows, which I quote verbatim:

''*Virchow's definition is too narrow, since it only includes adult tuberculous granulation. To this typical form must be added the young nodules visible only under the microscope and the irregular masses of cellular-embryonic tissue which have the same structure and the same fate as the tubercle and which are encountered either in acute granulia or in caseous pneumonias without granulation.*''

''*The form is not an absolute characteristic since the tubercle can present in the infiltrated form.*''

(1) *Grancher, On the Unity of Phthisis. Paris Thesis, 1873.*

Baumgarten, (1) who studied experimental pulmonary tuberculosis by injecting a bacillary emulsion into the anterior chamber of the eye and examining the pulmonary lesions at their different stages and at regular intervals, observed that bacilli attach themselves to the inter-alveolar septa and that they cause an inflammatory process there, itself causing the desquamation of the alveolar epithelium, which falls into the cavity of the alveolus. Subsequently, the tubercles are formed, the largest of which con- sist of a network of alveoli whose cavity is filled with epithelioid cells.

This conception of **Baumgarten** is not accepted by **Borel,** who criticizes it in these terms: **(2)**

''How do cells of such diverse origins (alveolar cells, pulmonary epithelium, vascular endothelium, lymphatic follicle cells, bronchial epithelium) manage to form the epithelioid cells of granulations? It is not very clear.''

''In reality, we will see how different pulmonary tuberculosis is and how an inoculation method (intravenous), which aims to bring bacilli and elements of lung tissue into immediate contact, will allow us to follow with the greatest rigor and day by day, the sequence of events that occur in pulmonary tuberculosis.''

''Infection of the lung by the venous route will have the advantage of showing us, with certainty and without trauma, the first stages of the infection, the immediate reaction of the organism, the rapid formation of true tuberculous granulations in the vessels themselves. These initial granules, true inoculation tubercles, will evolve for a certain time without causing any other apparent reaction and will result in caseification around the twentieth day.''

"At that moment, and without any transition, we will witness the rapid generalization of the tuberculous process."

From his observations, Borel concludes: *"The tuberculous cell is always a lymphatic cell."*

From the first day of injection, and even immediately after the injection, **Borel (3)** notes that: *''All the capillaries and vessels of the lung show intense polymorphonuclear leukocytosis. The clustering of leukocytes is especially evident in the places where the bacilli are found. At high magnification, it is easy to see that most of them contain bacilli. Sometimes, a small bacillary cluster is found stopped in a capillary of the lung; the mass of leukocytes arranged around it is often very considerable, and these form a barrier of several rows; they almost all contain bacilli... Given the constancy of the phenomenon, I consider it perfectly established that the bacilli introduced into the circulation are immediately apprehended by the polymorphonuclear leukocytes.''*

(1) *Baumgarten, Uber Tuberkel und Tuberkulose.*
(2) *A. Borel, Ann. de l'Inst. Past., p. 597, 1893.*
(3) *A. Borel, loc. cit., p. 604.*

This polymorphonuclear leukocytosis had already been observed by **Kostenich** and **Volkow (1),** who noted that these leukocytes are arranged in foci in which bacilli are almost always found, but which only affect neighborly relationships with the latter. They did not see leukocytes containing bacilli; but they noted that where there are bacilli, there are almost always polymorphonuclear leukocytes, and that the latter are good indicators of the presence of the former.

Borel noted that on the third day, the leukocytes begin to undergo a process of degeneration and disappear completely on the fifth day, which leads him to this conclusion (loc. cit. p. 606):

''The polymorphonuclear leukocytes are therefore powerless in the fight against the bacillus and are killed by it; but it is nonetheless evident that they incorporate the bacilli from the first hour and I cannot, in any way, subscribe to the ideas put forward by Messrs. Kostenich and Volkow.''

In the places where bacilli and polymorphonuclear leukocytes are found together, **Borel** notes the arrival, on the third day, of large cells with a single nucleus, presenting numerous expansions and which are, according to him, large mononuclear leukocytes.

They sit at the periphery of the vessels. He then notes that: *"From the third day, the sections show, in the very interior of the vessels, numerous giant cells and we see them, so to speak, forming around the bacillary clusters. Here, their origin is not doubtful; it is through the fusion of mononuclear leukocytes that they take birth."*

Furthermore, **Borel** notes *"the formation of similar giant cells, by the same process, in the alveoli, from the first days of inoculation."* But in this case, this formation occurs not by fusion of mononuclear leukocytes, but by fusion of cells called dust cells (which some authors consider to be epithelial cells). A drawing representing these cells is appended to **Borel's** memoir. They do indeed appear to be epithelial cells and their appearance differs completely from that of mononuclear leukocytes. Therefore, **Borel** strives to demonstrate, in order to preserve the unity of his demonstration, that these cells must be considered as cells of lymphatic origin.

In any case, these observations lead **Borel** to conclude that "the tuberculous cell is always a lymphatic cell."

This is also the conclusion of **Calmette (2)**, who considers that, in all cases, the tubercle is a lymphatic production and that *"the fixed cells of the different organs in which it is likely to develop play no active role in its histogenesis."*

(1) *Kostenich and Volkow, Development of the Experimental Tubercle. Arch. of Exp. Med., 1892, p. 741.*
(2) *A. Calmette, L'infection bacillaire et la tuberculeux, Masson, 1920, p. 104.*

Mr. and Mrs. **Hollande (1)** concluded from their cytology studies on experimental tuberculosis of the lungs of rabbits and guinea pigs:

"The histogenesis of the tuberculous follicle occurs at the expense of a single cell, the embryonic cell. This is simply a diapedetic lymphatic cell that will successively transform into a plasma cell, an epithelioid cell, and finally a giant cell.

This transformation is the result of the morbid processes that the cells undergo under the influence of microbial toxins and the endoparasitism of the tuberculosis virus."

Thus, we are faced with three different conceptions to explain the development of pulmonary tuberculosis.

The first, dating back to the time preceding the discovery of Koch's bacillus, makes pulmonary tuberculosis a consequence of chronic inflammation of the lung.

The second and third attribute tuberculosis to an infection of the organism by Koch's bacillus, but differ in the mode of development of the tuberculous matter.

The second, with **Grancher, Baumgarten,** etc., admits that the tubercle results from the desquammation, in the pulmonary alveolus, of the alveolar epithelial wall irritated by the tuberculosis virus, that is, from a prior pneumonic phenomenon.

The third, with **Koch, Cornil, Metschnikow, Yersin, Borel, Gilbert** and **Girode, Hollande,** etc., disputes this origin and considers that tuberculous tissue always arises exclusively from lymphatic cells that encounter the bacilli to engulf and destroy them.

The following objections can be raised against the second and third conceptions:

The second conception clearly explains how the presence of Koch's bacillus in the alveolar walls can, through inflammation, cause the alveolar epithelium to fall into the cavity of the alveolus and explains how the tubercle is initially formed. It does not explain how this tubercle grows or how the epithelial cells evolve, and even less what the small round cells or embryonic cells correspond to, which do not respond to any epithelial element of the alveolar wall and which, however, constitute the zone of growth of the tubercles and are visibly linked to this growth.

The third conception, according to which the tuberculous cell is always a lymphatic cell, can be opposed by the following:

1. If, at the beginning of the tuberculous infection, a polymorphonuclear leukocytosis of defense occurs, intended to encompass and destroy the bacilli, nature employs a singular defense process since these leukocytes are killed by the bacillus.

(1) *M. and Mme Hollande, Histogénèse et cytologie du follicule tuberculeux, Arch. de méd. exp., 1931, m. 149.*

2. Since, after the fifth day, there remain bacilli in the blood and also polymorphonuclear leukocytes in the body, these being, moreover, indefinitely replaceable by others of new formation, it is inadmissible that, if there is really a call for leukocytes by the bacilli, the polymorphonuclear leukocytosis does not continue. There is therefore a fact of great importance which remains unknown in the phenomenon.

3. If polymorphonuclear leukocytosis is a defense process, the mononuclear leukocytosis which follows it is also one; it is incomprehensible and even inadmissible that the tubercle bacillus kills the first leukocytes, polymorphonuclear cells, and that instead of killing the latter, it exerts on them an excitatory and hypertrophic action which causes them to transform into epithelioid cells or giant cells.

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4. The destruction of lung tissue results from the formation of tuberculous tissue, that is to say, the tubercle. Now the tubercle would be constituted solely by lymphatic cells. How can we admit that the tubercle bacillus, instead of directly carrying out the destruction of lung tissue itself, could carry it out indirectly by communicating to the lymphatic cells this destructive property which they do not normally possess. Now, this is indeed the action that the bacillus would exert according to **Borel's** conclusion: the tuberculous cell is always a lymphatic cell.

5. Proof of the inaccuracy of the third conception is provided by the fact that after the activation of a first defense process that would result in the death of the polymorphonuclear leukocytes, then after the activation of a second defense process that, on the contrary, would push the mononuclear leukocytes to a hypertrophic evolution, tuberculosis develops as well and even better than if no defense process had intervened, since it is precisely the mononuclear leukocytes that would constitute the tuberculous cells.

We realize that such assertions are based on facts whose interpretation is not exact and that they only respond to multiple hypotheses invoked each time for the needs of a thesis, to compensate for unknown facts and insufficient determinism.

In summary, neither one nor the other of the three conceptions in question explains the evolution of pulmonary tuberculosis, nor the nature of this disease.

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METHOD APPLIED TO RESEARCH

Since 1882, the date of the discovery of Koch's bacillus, the study of tuberculosis has not made any significant progress. It could not make any because an insurmountable barrier for observers was erected in front of them by a series of facts that had become intangible principles; it could not make any progress without this barrier being destroy-

ed by the development of new principles corresponding to the reality of the facts. I have indicated these principles on page 5. It is thanks to them and by subjecting every question to rigorous determinism that I have been able to fruitfully carry out this current study of the evolution of tuberculous lesions.

It is thanks to them that I was freed from false principles and was able to see the formation of the epithelial cells of the lung by a branch emanating from the carcass of the inter-alveolar septum, the constitution of these cells, the birth of embryonic cells on the carcass of the inter-alveolar septum or on bundles of fibrils that it emits, the real nature of these embryonic cells and finally the birth of Koch's bacillus on the granules formed by fully evolved embryonic cells.

Without the new knowledge described in my 1926 publication, I would have seen nothing, because the old knowledge would have forbidden me to believe that an embryonic cell can be born by a small filament itself coming from another, or to believe that Koch's bacillus, or any other bacillus, can be born on a granulation, and this especially because, not having been able to find another origin for them, it is generally accepted that embryonic cells are lymphatic cells coming from the blood by the hypothetical and probably non-existent phenomenon of diapedesis.

How can one believe, and even better dare to write, in the presence of current principles, that a granulation belonging to a cell, whether or not it is of lymphatic origin, can give rise to a Koch bacillus?

The hypothesis of determinism that was used in this study was excluded. I sought to establish the facts with the maximum possible precision and in such a way that they could be checked, appreciated, and judged directly by the reader without having to take into account the author's opinion.

Such a judgment is only possible on very clear, unretouched photographic documents, that is, on documents that reproduce fully, clearly, in the smallest details, the points of the preparations that served for the study and the determination of the conclusions.

This precision cannot be obtained by drawings, and to achieve it, it is absolutely necessary to abandon this reproduction process and replace it with photography, the only process capable of fidelity and which has the advantage of avoiding errors of interpretation by the eye and brain, omissions and drawing errors, as well as errors that may arise from the fact that, through his drawing, the author seeks to give a demonstration and that he reproduces above all the points which, according to his conception, are of a nature to make the demonstration he seeks.

Such a reproduction made with the eyes and the hand, always more or less misled by reasoning, *is necessarily inaccurate and open to question;* it is incapable of serving as a faithful witness to observations and proof of assertions. If the drawing is executed by the hand and ignorant eye of a draughtsman, inaccuracy is inevitable. A striking proof of this will be found in the following pages, by comparing the photographs of lung tubers, embryonic, epithelioid, or giant cells, and epithelial cells of the alveolar wall of the lung with the drawings that have been published on the same subject.

We will even realize, through this examination, that the exact reproduction of a microscopic preparation by a drawing is impossible, whatever the skill of the draftsman. The most tenuous details are sometimes the most important in a preparation, and an inaccurately placed line is enough to modify the relationship that exists between two elements. This results, quite obviously, from the comparison between drawings and photographs.

But photographic reproduction presents still other advantages. I have long noted that examining photographs of microscopic preparations under a magnifying glass allows for more fruitful observations than those made by direct examination under a microscope. Often, the examination of a photograph has allowed me to rectify an erroneous conclusion drawn from direct examination under a microscope, or to note an important fact that had escaped me during this examination.

When examining a preparation, a quantity of the most varied images passes before the eye, most of which are incomprehensible to the eye at the time, but which it may be able to understand later as soon as an understood image has opened the field to a new avenue of discovery. The sole subsequent examination of these images, fixed by photography and preserved, often allows one to make a discovery that had escaped direct microscopic examination.

Thus, I have long been led to definitively fix in photographs everything interesting that a preparation shows. I thus obtain and preserve a large quantity of faithful, precise documents, which, through their comparison and subsequent study, allow an extremely close analysis of the phenomena. The same region is often photographed at several different magnifications (100, 300, 600, 1200), to facilitate subsequent study.

If we add to these advantages that a drawing requires hours, sometimes a whole day, to execute, that it is expensive when it is executed by a draftsman, while obtaining a photographic image requires three to five minutes at most and that its cost price is minimal (0.80 to 1 franc at most), there can be no hesitation about which method to choose.

But, to apply the photographic method and preserve its character of perfect precision, it is necessary to reproduce only very clear, complete microscopic preparations, where the elements are visible with great sharpness and clarity.

The histological technique makes it possible to obtain very beautiful preparations in which the various elements can be stained differently to differentiate them. Very often, in these preparations, certain elements are barely visible and certain particularities, often of great importance, remain invisible. Such preparations are useful in certain cases, but, in general, they are not favorable for study.

Experience has shown me that, while it is very important to make the interior details of an element visible, *it is even more important to make the relationships between the elements very visible.*

Also, experience has led me to use almost exclusively coloured preparations, *stained in such a way that the elements all become visible, clear, and photographable.* The staining must be intense enough so that all the elements, whatever they may be, become visible and not so intense as to mask the interior details of the most massive elements. The question of the thickness of the section is also of particular importance; there is a thickness suitable for each type of study; very thin sections often present greater clarity, but a certain thickness is often necessary to be able to observe the relationships between the elements.

In this second volume, I have employed a process that I had used very little in the first, to present to the reader the results of the observations and the proofs of the facts; in most cases, I have carried out the photographic enlargement of the original photographs in the proportion of 1.7 to 2.5. This process has the advantage of:

1. It avoids the reader being obliged, in most cases, to take a magnifying glass to clearly distinguish the details; this does not prevent him from still having to add the magnifying glass research in certain cases, despite the enlargement.

2. It clearly exposes to the reader the finest details of the constitution of the tissues at a total magnification of 2000, for example.

3. It can present, with perfect sharpness of focus, very wide fields of observation, the original photograph having been taken at a low magnification.

I add that I used this enlargement process even for my personal observations. The study of the development of pulmonary tuberculosis, after injection of tuberculosis virus into the venous system, cannot show phenomena identical to those of the normal development of ordinary human tuberculosis.

The fact of inoculating tuberculosis through the venous route makes its development from the vessels obligatory. The observation, in this case, that the tuberculous cells first develop in the capillaries of the lung in no way leads to the conclusion that the beginning of the development of human pulmonary tuberculosis must occur by an identical process.

When studying experimental tuberculosis caused either by the venous route, or by inoculation into an organ, eye, liver, kidney, peritoneum, we can be certain of observing a mode of development which corresponds neither to that of pulmonary tuberculosis in humans, nor to clinical and natural tuberculosis in cattle.

It is therefore necessary, indispensable, to observe the normal or usual development of pulmonary tuberculosis, not to study experimental tuberculosis, which does not occur by natural means, and rather to study the natural and usual tuberculosis of humans.

The existence of a human tuberculosis bacillus, a bacillus of bovine origin, and a bacillus of avian origin has been recognized. We know that, even when developed in humans, the bovine bacillus retains its type, which can still be detected by certain characteristics; the same is true for the bacillus of avian origin. This at least allows us to think that the origin of these viruses is autogenous, that is to say, the bovine virus, for example, comes from the very organism of the ox, since it retains the specific characteristics of its origin. The human virus therefore only develops normally in humans, the bovine virus only in cattle, etc.

If the original source of the human bacillus is humans, it is therefore a mistake to study its development in rabbits or guinea pigs.

Only the experimental study of the development of the bovine bacillus in cattle, or of the avian bacillus in gallinaceous birds, would be exempt from this criticism, but the experimental introduction of the virus, by whatever means, would still constitute a cause of error for the observation.

Only the observation of clinical or accidental tuberculosis is free from these causes of error and can allow an exact analysis of the phenomena. It is for all these reasons that, in my studies, I have addressed pulmonary tuberculosis in humans. The anatomical pieces necessary for such studies are obtained at will during autopsies, in cases of pulmonary tuberculosis followed by death. It is very rare that one cannot find in the lung an area in which tuberculous lesions exist at all stages of their development, and in all their forms. A piece of this region is treated with 10% formalin for three days, or with Bouin's liquid, then washed, dehydrated with alcohol, then immersed in toluene, and finally embedded in paraffin. The sections, glued and deparaffinized, are then stai-

ned, cold, for 2 to 10 minutes, with Ziehl's acid fuchsin to which one-third, one-half, or three-fifths of its volume of alcohol is added, depending on the case;

The alcohol here fulfills the role of limiting the degree of staining. It is then washed with cold distilled water.

In other cases, the sections are first stained with ferric hematoxylin, then with Ziehl's fuchsin. These stains are sufficient to obtain preparations in which all the elements are visible.

The observations I am going to describe will consist, first, of examining a typical tubercle and analyzing the various elements that constitute it.

* * *

EXAMINATION OF THE ELEMENTS OF TUBERCULAR TISSUE

AND THE POINTS ON WHICH RESEARCH MUST BE CONCERNED

According to **A.-Ch. Hollande** and Mrs. **G. Hollande (1)**, who have extensively studied the cytology of tuberculous tissue, the tubercle is composed as follows:

''The tuberculous follicle comprises three zones: the center is occupied by the first diapedesic embryonic cells, transformed into epithelioid cells, more rarely into giant cells; the external zone contains the typical embryonic cells, recently diapedesic; the middle zone comprises the plasma cells and the epithelioid cells in formation.

There is no intermediate matrix between the cells of the follicle (p. 217)..."

In the same publication (p. 166), the same authors write:

''The tuberculous follicle therefore appears to have a very simple formation: it is the result of lymphatic production from the union of mobile, diapedesic cells, all of which originate from the same type of cell: the embryonic cell, itself a lymphoid cell.''

''In other words, in the same tuberculous follicle, the embryonic cells are simply found, to varying degrees, at different stages of their morbid development. Between these various cells, belonging specifically to the tuberculous follicle and especially to the periphery of this follicle, connective cells, fibroblasts, and polynucleated leukocytes will infiltrate, depending on the state of virulence of the microbe...''

''In a non-caseated tuberculous follicle, Koch's bacilli are never found in a free state; in other words, they are never located between the cells and outside of them; they are necessarily incorporated into the protoplasm of the epithelioid cells and giant cells, or into the protoplasm of the epithelioid or giant cells to collect the bacilli there.''

''Finally, let us note that there is no "intermediate gangue, special fibrils, reticular tissue" connecting the various cells of a tuberculous follicle.''

Let us now examine for ourselves a region of tuberculous tissue such as that represented in plate 1. This figure represents a section of a human tuberculous lung; it shows in the center a tuberculous nodule in the process of forming. This itself comprises a central mass ***b b b*** of compact tissue which barely stains with Ziehl fuchsin and in which we notice only a few scattered globular elements, of which we most often only perceive the outline.

(1) *A.-CH. Hollande and Mrs. G. Hollande, Histogenesis and Cytology of the Tuberculous Follicle, Arch. d'anal, microsc, 1931.*

Around this mass, we notice, in region ***f f***, a large zone teeming with embryonic cells and other larger ones which we have designated plasmocytes.

On another side of the young nodule, in ***c c***, we see almost no cells, and this entire region is occupied by long, branching, anastomosing fibers, which constitute a framework whose fibers will disappear into the central compact mass of the nodule, an arrangement notably visible in region ***a a***.

In ***k k***, then in ***i i***, are two other regions in which we find embryonic cells, plasma cells, and a network of branching fibers.

In ***d*** and ***e***, outside the follicle, we notice two giant cells with their nuclei and protoplasmic expansions.

Finally, in ***y*** and ***j,*** we see larger elements which are the epithelial cells of the alveolar walls; in ***g,*** a small part of the alveolar cavity still persists.

If we examine other regions of the same preparation, those where we only see infiltrating tissue, without nodular forms, we see that the tuberculous tissue is made up of the same elements.

Plate 1 bis shows another tuberculous follicle, more completely developed and rounded in shape, at the periphery of which we notice, in ***d, e, f,*** the same tracts or branching fibers, anastomosed already seen at the periphery of the follicle in plate 1.

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Here, these fibers develop parallel to the outer circumference of the follicle and appear to constitute a sheath or fibrous envelope, which has been considered, for this reason, as a fibrous reaction intended to encyst the tubercle; these fibers do not, in reality, have this purpose. It is noted that the closer they are to the central mass of the tubercle, the thicker they are and the less power they have to fix Ziehl's fuchsin. In fact, these fibers or tracts evolve, increase in thickness by multiplication of the elements that constitute them and they end up forming a compact substance similar to that which forms the center of the tubercle, the empty, aerolar spaces that exist between the fibers gradually fading. This transformation is clearly observed in region ***b, b, b,*** where it took place more rapidly than in regions ***d, e, f.***

In ***g, h,*** we see these tracts heading in another direction, which shows that their function is not to encyst the nodule. In ***l, m***, we see an inter-alveolar septum considerably thickened by tuberculous tissue that has infiltrated it. In ***k,*** at the periphery of the nodule, we see a region rich in embryonic cells.

Pl. 1. *Section of tuberculous lung. Magnification 110. Enlargement to 200.*
Pl. 1 bis. *Section of tuberculous lung. Magn. 110. Agr. At 200.*

In the two large cavities at the bottom of the figure, we notice large globular elements that are epithelial cells of the alveolar walls that have fallen into the cavities.

These two **plates 1 and 1 bis** therefore clearly show us that the main elements that constitute tuberculous tissue, whether in the nodular or diffuse form, are the following:

1. Embryonic cells and plasma cells.

2. Epithelial cells of the alveolar wall.

3. The intermediate matrix between the giant cells, formed by the branched and anastomosing tracts or fibers.

4. The giant cells.

We will study successively the nature, origin, and evolution of each of these elements.

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ORIGIN AND NATURE OF EMBRYONIC CELLS

The nature and origin of embryonic cells are currently unknown. They have most often been considered to be lymphatic cells that have emerged from the vessels by diapedesis because no other origin could be found for them, and this phenomenon has been explained by a special action of the toxins of Koch's bacillus on these cells; they would emerge from the vessels by diapedesis to destroy Koch's bacillus on the spot; this would be a phenomenon of the body's defense.

I sought the origin of these embryonic cells in the regions where they exist or appear in greatest numbers, that is, at the limit of the extension of the tuberculous infiltration and at the periphery of the growing tubercles.

This study was carried out on sections of fragments of human tuberculous lung taken as far as possible at the limit of the tuberculous lesions and in regions rich in miliary tubercles. These fragments were fixed in a 10% formalin solution or in Bouin's liquid, embedded in paraffin by ordinary methods, and cut into sections of 1/200 to 1/400. The sections were first treated with hemalun, then stained with varying intensity, depending on the case, with Ziehl's carbolic fuchsin. To moderate the coloring power of this reagent, a little 95° alcohol is added in the proportion of one-quarter to one-third. In some cases, the sections were stained with carbolic fuchsin alone.

By studying the embryonic cells at length one by one, in the regions where they abound, I observed:

1. That many embryonic cells are not round, as described, but are oval or pyriform because they generally have one side more pointed than the other.

2. That a rod of varying thickness and length is adhered to this more pointed side.

3. That this stem, which is the pedicle of the cell, starts or often seems to start from a fairly long neighboring branch from which it emanates.

By multiplying this observation hundreds of times and photographing the observed arrangements each time, I had in hand a mass of facts which proved in a certain, indisputable way that embryonic cells are cells formed at the end of a pedicle itself born on a branch coming from an undetermined region. The formation of an embryonic cell can be compared to that of a plum or a cherry whose tail or peduncle starts on a small branch.

When one has devoted oneself to this observation for a very short time, one can perceive the pedicle of most of the embryonic cells of a region, if the coloring of the section has been suitable for this purpose.

Plate 2 represents a point in a region of lung undergoing infiltration. This region was photographed at a magnification of 1100 and the image enlarged to give a magnification of approximately 2000. A bundle ***xxx*** composed of fibrils gives rise to the embryonic cells: ***b,*** slightly stained, whose pedicle is at ***b'; c,*** whose pedicle is at ***c',*** then to the other cells ***j, k, l, M, K, L, u, q, r, v', s, x',*** also connected to the bundle ***x*** by fairly clearly visible pedicles.

Note that the pedicles ***b', c', G*** of the embryonic cells ***b, c, F*** arise very clearly from a large refractile granulation ***b", c", H*** placed in the bundle ***x.***

The pedicle ***j'*** of cell ***j*** also arises from a large granulation ***j"***.

At ***e d, g, i,*** there is a group of tightly packed embryonic cells whose pedicles are directed, one towards the top of the figure, the others downward, and four of which, ***f, f, g, i,*** start from bundle ***xx***.

A group of 7 or 8 embryonic cells exists at ***y, N, P, D, R, a,*** packed against each other. The pedicle of the large cell ***y*** exists at ***S.*** The pedicle of the large cell ***a*** is located at ***a'*** and starts from a small bundle ***A*** at the same point as four other branches ***B, C, D, E,*** which also form the pedicles of other cells, two of which are marked with a cross.

Another embryonic cell is seen at ***m*** with its pedicle ***m',*** another large one at ***n*** with its pedicle ***n'*** which is a ramification of the branch ***n" n"*** starting from the bundle of fibrils ***x x.***

Other very small cells of different sizes, with a fairly clear pedicle, are seen in the ***z z*** regions, particularly at the very bottom of the figure, and it is noteworthy, particularly

for the four very young cells ***z'*** and ***s",*** at the bottom of the figure, that they are born by a short filament on a large granulation and that the combination of the two granules and the rod that connects them has the appearance of a dumbbell.

To the right and at the bottom of the figure, we will see a young embryonic cell ***q*** born by the rod ***b,*** on a small granulation ***10.***

Plate 2 shows, moreover, that between the embryonic cells a network of branched fibers has developed such as ***xxx*** and its lateral branches, those of the ***z*** regions, and those on which the young embryonic cells ***z' z"***... etc. are born.

The figure contains other very interesting particularities, but for the moment, we will limit ourselves to pointing out that it demonstrates:

1. That embryonic cells are not isolated, free cells.

2. That these cells are born at the end of more or less long pedicles emanating from bundles of branched fibrils, and that these pedicles are themselves born on one or more granulations included in the bundles.

3. That they remain linked to these bundles throughout their development since we see them in the state of large adult cells such as ***y, a, b, n,*** of medium-sized cells such as ***C, j, k, m,*** or smaller ones, such as ***V,*** then ***T, T',*** and finally of tiny cells which are still only a large granulation such as ***z', z"***.

4. That, consequently, being born and developed on the spot, they do not come from the blood by diapedesis and have nothing in common with lymphatic cells.

This demonstration is carried out numerous times in most of the plates of this work, without having been specifically sought.

In Figure 1, **plate 3**, we will see, mainly, a group of embryonic cells ***a, b, c,*** emerging on a ramified branch ***d,*** as well as other scattered embryonic cells provided with pedicles, some of which ***e, f, g,*** are characteristic, their end being terminated by a small spherical ball. We will see later that this small ball is one of the two granulations of a dumbbell-shaped rod which gave rise to the cell; the second ball of this dumbbell-shaped rod is located in the cell and at the periphery.

In Figure 2, **plate 3**, we can distinguish a branch ***b,*** divaricate, emitting secondary branches ***c, f, g, h, i, j.*** An almost sessile embryonic cell ***n*** is born in ***e***; others are born on the branches at ***l, m, A, k;*** other embryonic cells whose pedicle or point of attachment is seen are seen at ***r, w, v, y, z***.

Pl. 2*. Section of tubercular lung. Magnification 1,100. Enlargement to 2,000.*

Let us also note here that the branches such as ***a, d, b, t', p, u,*** and their ramifications constitute the gangue or intermediate framework which will be discussed later. This figure clearly demonstrates the existence of this gangue and the formation, by it, of embryonic cells.

Plates 4 and 5 show in two other cases of pulmonary tuberculosis, two different aspects taken by the fine ramified branches which give rise to the embryonic cells, in the regions where the latter abound in large areas, that is to say in the zone of extension of the tuberculous infiltration.

In **Plate 4,** Figure 1 shows at ***a, b, c, e, f, u, r, s, t, z***, embryonic cells with their pedicles; at ***g,*** a sessile embryonic cell on a branch; at ***h,*** a branch giving rise to eight embryonic cells; at ***i,*** another branch also giving rise to eight shortly pedunculated cells; at ***j,*** another branch giving rise to a large cluster of cells. Other large clusters of embryonic cells are also seen at ***f, v, x, y.*** Cluster ***x*** arises on a branch emanating from a larger branch ***m***; at ***p,*** we see a large branch showing the birth of small, highly branched lateral branches such as ***q,*** which give rise to embryonic cells. It is a series of small lateral branches similar to ***q*** and brought together on a branch, or terminating it, which give rise to clusters such as those in regions ***f, v, j.*** At ***n,*** we see a divaricated branch, with the same destination as branch ***b,*** figure 2, plate 3, which gave rise, at each angular projection, to a short peduncle carrying an embryonic cell.

Figure 2, **Plate 4** represents a region immediately adjacent to the previous one. It shows, like Figure 1, by filaments visible everywhere, the first elements which form the gangue or intermediate framework. It is these filaments which give rise to the embryonic cells which abound in very extensive areas. In this figure we will see pedunculated embryonic cells, in ***a, b, g, h, i, j, k, l, m.***

Plate 5 shows a region of a tuberculous lung where the tuberculous tissue already comprises a gangue of filaments ***a, b, c, d, e,*** etc. more developed than in the previous figures;

These filaments give rise to numerous embryonic cells of all sizes, from the smallest to the largest. As an example, in ***A*** and ***B,*** we see embryonic cells joined by their pedicle to their formative filament; we see smaller pedunculated cells in ***K, E;*** other pedunculated embryonic cells of various sizes are visible at the points marked with a cross.

At the bottom of the figure, we see a giant cell which will be described later.

Pl. 3. *Section of tuberculous lung. Fig. 1. Magnification 550. Enlargement to 1,000. Fig. 2. Magnification 1,100. Enlargement 2,000.*

*At the top, the reduced cavities of the pulmonary alveoli still exist, and we note, a fact of the greatest importance, the penetration, in **f,** for example, of the filaments of the gangue or intermediate framework into the interalveolar partitions which they thicken and where they also form embryonic cells.* This is the picture and the mechanism of the infiltration of tuberculous tissue into the still healthy regions and of the progression of tuberculous lesions from one place to another.

By carefully examining the embryonic cells in **plate 6**, one after the other, with a magnifying glass, we can see for most of them the pedicle that formed them and, sometimes, the point of attachment of this pedicle to the web of filaments that covers the figure.

We will examine especially, from this point of view, the cells ***a, b, c, f, g, j, m, n, o, q, s, t, u, v, x,*** as well as all the cells marked with a cross; in ***r,*** we will see three smaller, pedunculated cells in the process of growth; in all the points marked with the letter ***i,*** we will see young pedunculated cells of various sizes in the process of formation.

The filaments that form the web on which the embryonic cells are born stain much less easily than the latter and are no longer acid-fast, in general.

Plate 6 alone contains the demonstration of the nature and origin of embryonic cells; it shows that these cells are born at the end of a filament, by a small granulation which develops on the spot until the adult state of the cell. **Plate 6** also gives rise to an interesting observation.

Plates 4 and 6 present a characteristic difference in appearance which corresponds to the age, that is to say to the degree of evolution of the tuberculous tissue; in plate 4, the few branches give rise to a considerable number of embryonic cells which are of entirely new formation and of which none are yet in the process of germination. In plate 6, the branches are excessively numerous, very ramified and bear a considerable number of young, tiny, pedunculated embryonic cells, some of them very colored, most very lightly; this network of fine branching filaments comes from the germination of the oldest embryonic cells; the demonstration of this origin will be made several times subsequently.

The proof is therefore given by the figures in plates 2, 3, 4, 5, and 6 that, in the regions where embryonic cells abound, they are born at the end of a formative pedicle, a thin filament which, itself, originates on another filament. I will not insist further on this demonstration, because it will be repeated and visible in most of the plates which follow.

Pl. 4. *Section of tuberculous lung. Gross. 335. Agr. 600.* ***Pl. 5.*** *Section of tuberculous lung. Gross. 335. Agr. 600.*
Pl. 6. *Section of tuberculous lung. Gross 535. Agr. 600.*

The origin and nature of embryonic cells is therefore proven at once:

1. By the fact that they are located at the end of a formative pedicle, itself emanating from a long filament which gives rise to a series of cells.

2. By the fact that they are born by a small pedunculated (or sessile) granulation and grow on the spot to gradually reach the size of adult embryonic cells.

This mode of birth and development, which establishes the origin of embryonic cells, therefore demonstrates that they do not come from the blood by diapedesis and that they are not and cannot be lymphatic cells.

However, for the demonstration I have just given to be complete, it remains to determine the origin, the starting point, and the nature of the filaments that form the embryonic cells in the regions where they abound.

It is easy to see, by examining pulmonary tissue, in the intermediate region between healthy tissue and tuberculous tissue, that the first sign that appears in the barely affected inter-alveolar septa is the formation of embryonic cells.

I therefore sought to determine where these embryonic cells come from, either in the barely affected inter-alveolar septa, or in the septa in the process of destruction and which are losing or have lost their epithelium.

This study allowed me to discover:

1. The nature, origin, and real role of embryonic cells. We have just demonstrated that they are not lymphatic cells and that they are formed on site by certain filaments; but their nature and role still remain unknown to us.

2. That embryonic cells germinate and themselves emit the filaments that form the large areas where embryonic cells abound.

I was necessarily led, during this research, to study the nature and evolution of desquamated cells in the inter-alveolar cavities.

The following research will therefore have as its object:

1. The origin of the first embryonic cells formed in the interalveolar septa and the role of these septa in their formation.
2. The emission of fibers and fiber bundles by the inter-alveolar septum.
3. The nature and evolution of desquamated cells in the pulmonary alveoli.

Later, our study will continue with that of giant cells, then with that of the gangue or intermediate framework.

ROLE OF THE INTER-ALVEOLAR SEPARATOR

IN THE FORMATION OF EMBRYONIC CELLS

The exposition of this study will not be long, because the demonstrations consist of facts that are materially fixed by photography and, since the reader's conviction must be quickly achieved by examining the photographs alone, a few words of explanation will suffice for each plate.

Plate 7 represents a section of tuberculous lung in the immediate vicinity of extensive infiltration lesions; it shows a group of disorganized pulmonary alveoli and inter-alveolar septa. The section has reached some septa perpendicular to their direction and others more or less obliquely.

The epithelial cells have desquamated with the exception of a single one ***A***, still connected to a wall; only two or three cells ***B, G, D*** remain in the alveolar cavities, which are empty.

We note here that all that remains of the inter-alveolar septum is a carcass completely empty of both its epithelium and its capillary vessels. The word carcass corresponds very exactly, in fact, to what remains of the septum; moreover, these alveolar septa are completely free of elements foreign to them, notably those of the neighboring tuberculous infiltration tissue. However, they are already rich in embryonic cells. By carefully examining all the points of the septa, we see that all these embryonic cells were born on the carcass of the wall. Some are sessile, others provided with characteristic pedicles. They can be examined at all the points marked by letters and there are others not designated. We will see sessile embryonic cells in ***h, p, r, t, u, g, i, j, k, s, o, v***.

Here, the birth of embryonic cells on the wall is evident, certain. Thus, we observe, in fact:

1. That, the inter-alveolar septum is affected, has lost its epithelium and its blood capillaries.

2. That, it has become covered with embryonic cells born on its own elements.

3. That, these septa are not diseased because they have been invaded or infiltrated by neighboring tuberculous tissue.

Pl. 7*. Section of tuberculous lung. Gross 335. Agr. 600.*

These facts are easily explained by the disappearance of the blood capillary system. The total cessation of circulation in a region must obviously determine its disorganization and subsequent necrosis.

As for the formation of embryonic cells on the wall, it appears to be the expression of a normal physiological phenomenon: the repair of the pulmonary epithelium when it has been damaged. It is likely the embryonic cells that are the starting point for the regeneration of epithelial cells when this repair must take place, as in congestive pulmonary conditions, such as pneumonia.

Plate 7 shows that, even when this epithelial repair does not take place, it is still initiated by the formation of embryonic cells. In this case, it is necessary to determine the subsequent evolution of these cells that do not fulfil their role as regenerators of the epithelium. This evolution is obviously that of embryonic cells, in general, regardless of where they are formed.

Summarizing the preceding observations, we see that, when a region of pulmonary tissue must be destroyed to be replaced by tuberculous tissue, it is the site of three phenomena:

1st, the destruction of the network of blood capillaries;
2nd, the destruction of the epithelium whose cells fall into the alveolar cavities;
3rd, the formation of embryonic cells on what remains of the inter-alveolar septum, which I call the carcass of this septum.

It is likely that the destruction of the network of capillaries is the first phenomenon to appear in chronological order and which determines the dissociation and fall of the epithelium.

Let us further complete the demonstration already made by other documents.

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Plate 8 shows an inter-alveolar partition ***A, B, C, D,*** in very slightly oblique section, to which was joined another partition ***E, F, G,*** which appears to have been cut almost in its own plane; two alveolar cavities, ***H*** and ***K,*** contain various categories of elements.

Let us first study the inter-alveolar septum ***A, B, C, D***. It still shows in ***G*** an epithelial cell not yet eliminated and a carcass in the process of disorganization which appears quite free of foreign elements coming from the already tuberculized regions.

However, neither the epithelium nor the capillaries no longer exist, and this is evidently for a reason unrelated to the action of the tuberculous infiltrating elements since none are seen.

Pl. 8. *Section of tuberculous lung, Gross. 335. Agr. 600.*

Nevertheless, and despite the absence of these infiltrating elements, the carcass of the septum gives rise to numerous embryonic cells, some sessile in ***b, h, o, p, z,*** others shortly pedunculated in ***i, j, m, n, q, u, s.***

These facts therefore lead to the same conclusions as for Plate 7.

Plate 8 also shows another important fact; it is, in ***K,*** a group of very fine, branched filaments that come from the group of embryonic cells ***L, L, N*** and are formed by them. These are filaments emitted by the phenomenon of germination of embryonic cells; the latter evolve and, at the end of their evolution, have formed granules that germinate and each emit a filament.

The constitution, evolution, and germination of embryonic cells are studied further, and the phenomena that I briefly indicate here, since they are present before our eyes, will be the subject of very complete demonstrations.

Plate 9 gives a third and final demonstration of the existence of the alveolar carcass that remains after elimination of the epithelium and blood capillaries **(1)** and of the birth of embryonic cells on this carcass.

Figure 1 of this plate represents the photograph of a region close to that shown in plate 7 and therefore belongs to the same case of tuberculosis; it shows several inter-alveolar septa ***A, B, G,*** cut more or less obliquely and another ***D*** cut perpendicular to its plane.

The partitions ***A, B, C***, and ***D*** are now formed only by the carcass that remains after the elimination of the epithelial cells and capillaries. This carcass gives rise to numerous embryonic cells, a certain number of which are marked and designated by letters in the figure.

The inter-alveolar partition ***D*** shows, in the space between the letters ***p*** and ***s,*** the residual carcass in one of its most characteristic aspects, with the bundles of fibrils that form it and limit the cavities that will be occupied by capillaries or epithelial cells; on this carcass ***DD,*** we see, as on that of the other partitions, the formation of embryonic cells ***m, n, o, p, s.***

Figure 2 of **Plate 9** represents a fragment of disintegrated inter-alveolar septum, showing the birth on the residual carcass of this septum, of vegetative branches **such** as ***m, n,*** and of young tiny embryonic cells in ***o, x, y, q, w, e***..., etc.

(1) *Or rather perhaps of the epithelium of the capillaries, because the wall of these appears to be constituted only by the epithelium which will be described later.*
Pl. 9. *Section of tuberculous lung. Fig. 1. Gross 335. Agr. 600. Fig. 2. Gross 1100, Agr. 2,000*

Plate 10 shows an infiltrated and thickened inter-alveolar septum ***A, B, G, D, E, F,*** cut obliquely, and another portion ***T, R, S, H, K,*** cut at least partly in the plane of the epithelial surface, because the remains of a certain number of these cells can still be seen in ***K, H, L, k, P***, for example. By carefully examining the entire left-hand region of the figure with a magnifying glass, one will see the formation of embryonic cells of all sizes in ***l, m, n, o, p, q, r, s, t, u,*** and one will notice that a certain number are born on the residual carcass of the epithelial cells. One will also see embryonic cells with their club-shaped pedicle in the inter-alveolar septum ***A, D, E***, in ***a*** and ***c,*** for example.

Thus, the embryonic cells observed in the inter-alveolar septa can have two origins; they can be formed directly by the supporting framework of the inter-alveolar septa, or by filaments foreign to this septa and coming from neighboring regions of infiltration. It is only at the very beginning of tuberculosis that these cells have only one origin: the supporting framework of the inter-alveolar septa.

Thus, this fact of capital importance is demonstrated: the inter-alveolar septa, in the process of degeneration, and which has lost its epithelium and its circulatory system, gives rise on the framework which still persists, to a large number of epithelial cells. This framework constitutes, very probably, *the supporting organ* of the inter-alveolar septa and, moreover, the generative part of the epithelium (the fact will be demonstrated further on). It is clear that this formation of embryonic cells is intended *for the regeneration* of the epithelium.

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Following congestive lesions of the lung, two cases may arise:

1. in an ordinary pneumonic lesion followed by healing, the lesions of the epithelium, accompanied by lesions of the capillaries, are repaired by budding of the supporting carcass and formation of embryonic cells which reconstitute the destroyed elements;

2. in a pneumonic lesion which does not heal, budding of the supporting carcass also occurs, but the embryonic cells thus formed, not having fulfilled their role of replacing the destroyed cells, follow another evolution. Let us say immediately that *it is this evolution which constitutes pulmonary tuberculosis and which is its starting point.*

Before studying this evolution, we still have to complete our determinism, which is insufficient.

I have just demonstrated that embryonic cells are born and formed on the supporting carcass of the inter-alveolar septum. It is obviously by this process that the very first embryonic cells of the onset of tuberculosis are formed.

Pl. 10. *Section of tuberculous lung. Grosst 335. Agr. 600.*

But I have also previously demonstrated that, around the nodular formations and in the infiltration tissue, the embryonic cells, which form in considerable quantities, originate on filaments whose origin and nature are undetermined. It is this nature and origin that we must seek.

We saw above, regarding the explanation of **Plate 8**, that embryonic cells germinate and give rise to very fine filaments. I do not wish to develop this point here, because it must be developed in its proper place and in connection with the evolution of embryonic cells. Let us simply say here that several of these filaments, united together, give rise to the larger ones on which new embryonic cells will form. A single one of these very fine filaments can also give rise to a cell. It is therefore the first embryonic cells formed on the inter-alveolar septum which, by their germination, give rise to the first filaments whose union forms the branches or bundles on which new embryonic cells will develop.

This is the origin of the formation of the filaments which, either in nodular formations or in diffuse infiltration tissue, constitute a highly developed network which is the matrix or intermediate framework and on which new embryonic cells forms.

This origin is not the only one. There is another which is the supporting framework of the inter-alveolar partitions; this, just as it forms embryonic cells, emits fibers and fibrils which will branch out far and wide; we will demonstrate this.

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VEGETATION OF THE SUPPORTING CARCASS

INTERALVEOLAR PARTITIONS IN THE PROCESS OF DESTRUCTION.

EMISSION OF FIBERS AND BUNDLES.

We will see later, regarding the organization of the cells of the alveolar epithelium and particularly in **plates 17, 18, and 19**, that these cells are formed by branches emitted by the supporting carcass of the inter-alveolar partition. This makes us understand that the embryonic cells that are born on the supporting carcass of the inter-alveolar partition in the process of destruction have the function of reconstituting the cells of the destroyed epithelium. Indeed, we see that, when they become disorganized, they are formed by one or more branches emanating from the supporting carcass.

By carefully examining the remains of the inter-alveolar septa that are quite frequently encountered in the infiltrating tissue or at the periphery of nodular formations, it can be seen that these remains emit branches that contribute to the formation of the infiltrating tissue.

Plate 11 represents the photograph of a section of lung taken in a region of tuberculous infiltration, in a case of pulmonary tuberculosis in man. It shows in A, A, A, a fragment of

inter-alveolar septum emitting the branches Q, M, M, R, L, K, B, C, D, E, P, G, which anastomose with other filaments and constitute the neighboring infiltration tissue. These branches give rise to embryonic cells; in a, we see one of very characteristic shape, equipped with its club-shaped pedicle. **Plate 11** provides, moreover, a beautiful demonstration of the origin of embryonic cells. We will see pedunculated cells in all the places marked by a cross; some are sessile or almost, others are equipped with a more or less long pedicle and which, for some, takes the characteristic club-like shapes (***g, h, i, j, k***).

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We will notice, at many points, small pedunculated embryonic cells of various sizes in the process of formation; we see three side by side in ***b,*** others in the vicinity in ***d, e, f,*** then higher in ***o, p, q.***

Plate 12 represents a photograph taken in a region of infiltration of the same preparation. It is also very demonstrative. It shows an alveolar cavity containing debris of desquamated and degenerated epithelial cells, ***O, P, Q, R, S, T, U, V;*** this cavity is still limited quite clearly by a remnant of the carcass of the inter-alveolar septum cut obliquely at ***A, B, E,*** and of which only debris remains at ***J, M, N***. At certain points, this region ***Q, Q,*** was adherent to the edge ***G*** of the inter-alveolar sep- tum, as it still is in ***B,*** but it was detached by the preparation maneuvers and probably by the razor. From the septum branched branches, such as ***a,*** for example, which rami- fied into the interior of the alveolar cavity where they formed a lascis in which a few epithelial cells were imprisoned. On this lascis of ramifications, numerous embryonic cells of varying size were born in the process of formation, ***b, c, d, e, f, g, h, i, j;*** one will see a large one of typical shape, at ***k,*** with its nascent pedicle on a piece of the frame- work ***k'*** still not destroyed by an epithelial cell.

Another epithelial cell ***M*** which is still adherent to the alveolar carcass ***L,*** has formed at its periphery an embryonic cell ***l'***. We see numerous other embryonic cells formed on the residual branches of the carcass, or on the branches emitted by its vegetation, and in this case, the embryonic cells are sessile or very clearly pedunculated. We will see them in ***m, n, o, q, s, t, v, x,*** and in all the places marked by a cross.

A young embryonic cell, ***y,*** is born very clearly by a pedicle of characteristic shape on the frame of the degenerating cell ***R.*** Other tiny embryonic cells in the process of forming, pedunculated and very clearly, exist in the places marked by the letters ***p, r, u, y, z,*** and in numerous unmarked places.

This photograph therefore shows with great clarity what becomes of the supporting carcass of the partitions of the pulmonary alveoli and the degenerating epithelial cells;

Pl. 11. *Section of tuberculous lung. Gross 335. Agr. 600.*
Pl. 12. *Section of tuberculous lung. Gross 335. Agr. 600.*

it shows that this carcass vegetates and emits branches which ramify; it shows, moreover, that all these elements, supporting carcass and debris of epithelial cells give rise to embryonic cells, that is to say that, although losing their characteristic organized form, they remain alive and that the living matter which constitutes them continues to manifest its activity by the formation of branches and embryonic cells.

The same is true of the elements which constitute the wall of the vessels.

Plate 13 represents a photograph of a section of tuberculous lung taken in the very plane of a septum. Several capillary vessels, ***BC, EFG***, are cut longitudinally; the wall of one of them, ***B,*** rises to ***m*** where it gives rise to two pedunculated embryonic cells ***k'*** and ***f.*** The wall of vessel ***A*** also emits a short branch ***a'*** which divides into two others, each giving rise to an embryonic cell ***a*** and ***b.***

Also note in this figure 1 other pedunculated embryonic cells, ***d, e, g, l, o, p, t, u, v.*** It may seem surprising to the reader that pieces of alveolar septum and degenerated, fragmented, anatomically dead epithelial cells continue to vegetate and can give rise to embryonic cells.

If we examine, at very high magnification, the elementary constitution of the carcass of the inter-alveolar septum, as well as that of the desquamated and degenerating epithelial cells, we see that they appear to be made up mainly of two types of elements:

1. Short rods bearing a ball at each end, the dumbbell rods, which form almost all of the elements.

2. Granulations emitting a divaricate filament bearing 3 or 4 very short-pedunculated granulations of 2 to 3 m.

These are the elements that remain alive and continue to evolve; the organization of the inter-alveolar septum and the epithelial cells is dead, but the fundamental elements that constitute them remain alive and continue to evolve by forming embryonic cells. In fact, the demonstration of the persistence of life in the fundamental elements is given by the formation on the residual carcass of degenerated epithelial cells, of embryonic cells at all stages, from the smallest to adult cells.

As for the elementary constitution of epithelial and embryonic cells, it will be found exposed in various chapters that follow.

It must therefore be concluded, following the facts highlighted in plates 10, 11, 12, 13:

Pl. 13. *Section of tuberculous lung. Gross 335. Agr. 600.*

1. That the carcass and fragments of the degraded and degenerating inter-alveolar septum emit branches capable of forming embryonic cells.

2. That the residual fragments of desquamated and degenerated epithelial cells also give rise to embryonic cells.

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STUDY OF THE CONTENTS OF THE LUNG ALVEOLI

UNDER TUBERCULARIZATION

This content is made up, first, of desquamated epithelial cells, then of embryonic cells, and finally, at least at a certain point, of a network of filaments.

From his study of experimental tuberculosis caused by an injection of bacilli, **Baumgarten (1)** concludes that the latter attach themselves to the inter-alveolar septa and there cause an inflammatory process that causes desquamation of the alveolar epithelium; he states that, without any possible doubt, the large cells seen in the pulmonary alveolus are epithelial cells. Later, he says, the tubercles are formed, the largest of which are formed from a network of alveoli whose cavity is filled with epithelial cells.

Thus, in experimental tuberculosis, the tubercle would be formed by desquamated epithelial cells, according to **Baumgarten**.

Borel, who also studied the development of experimental tuberculosis in rabbits, disputes that the tubercle could have this origin, and he sought, after **Yersin**, to demonstrate that the tubercle always arises from a lymphatic cell. He had to note, however, that certain elements of the alveolar wall, the dust cells, very particular elements, he said, can form giant cells. But, after various considerations and by induction, he arrives at admitting that these elements must be included among the elements of lymphatic origin.

Currently, some still dispute that the large cells contained in the pulmonary alveoli are epithelial cells.

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When we examine the lesions of a tuberculous lung at a fairly low magnification and at their limit of extension, we see that it is always at this limit only, and not much further, that cells come to accumulate in the alveoli; we note moreover that, when an alveolus contains large free cells in its cavity, the inter-alveolar septum is in the process of disorganization or in the process of invasion by the neighboring infiltrating tuberculous tissue.

(1) *Baumgarten, Uber Tuberkel und Tuberculosen, Berlin, 1885; Exp. und palhol. Anat. Unter-suchungen uber Tuberkulose. Zeitschr. f. Klin, Medicin. Banv. IX and X, 1890.*

This, is already a fairly clear indication that the free cells in the alveolus are those of the epithelium, fallen into the cavity. This presumption, if it is not sufficient, is replaced by a certainty when we study their constitution and their relations with the alveolar wall.

This study, to which we are going to devote ourselves, will also provide us with new knowledge on cellular constitution in general and these will be of great help to us for the continuation of subsequent studies.

In the slow and progressive spread of tuberculous lesions, it seemed to me that it is above all, and perhaps always, through infiltration of neighboring tuberculous tissue that the alveolar walls are affected during successive attacks that destroy the pulmonary tissue. The alteration produced by this infiltration in the inter-alveolar septum is immediately visible and is accompanied by the appearance, in the alveolar cavity, of various types of elements.

The nature of these is not always the same, which is easily explained if we consider:

1. that the alteration of the alveolus is more or less old and that the nature of the elements that appear in its cavity is directly related to this age.

2. that elements that appear in the cavity may not remain there, being expelled outside by expectoration if the permeability of the bronchioles has not disappeared.

We may encounter in the alveolus:

1st either exclusively large cells, of various sizes;

2nd either exclusively small round cells, embryonic cells, which is rare;

3rd either large cells in the process of degeneration, accompanied by a certain number of embryonic cells in various stages of development, the most frequent case; often, in addition, these elements are accompanied by branched filaments;

4th either a vegetation of very branched filaments forming a network more or less tight, sometimes very dense, encompassing in its meshes some large cells that are more or less recognizable and embryonic cells in various stages of development.

This fourth case will be studied in connection with the development of the tubercle of intra-alveolar origin.

We see from this enumeration that what must concern us here is the nature of the large cells that have fallen into the alveolar cavity and their evolution, then the embryonic cells that are mixed with them.

CONSTITUTION AND NATURE OF THE LARGE CELLS

There is an easy method to prove, beyond any doubt, that these large cells are the normal epithelial cells of the wall, desquamated in the alveolus under the influence of the disorganizing cause that is the infiltration of tuberculous tissue: it is to look in the preparations for the points where the section has affected the inter-alveolar septum in the plane of the epithelial surface, while the elements of the latter are not yet completely detached and to photograph them in this state and with the relationships they have between them; then to compare these elements with those which are already desquamated in a neighboring region.

I point out, once and for all, that all the figures in **plates 15 to 20** are photographs of sections of tuberculous human lungs and represent regions where the lesions are in the process of spreading.

Plates 14 and 15 (Fig. 1 only) show elements still adhering to the alveolar wall; plates 17 and following show photographs of desquamated epithelial cells.

In plate 14, we see in ***A.D.E.F.B***. what remains of two inter-alveolar partitions cut more or less obliquely. In region ***D.E.F***., there is a mass of epithelial cells that have retained their relationships to each other. Examining them, we are led to the following observations:

1. The epithelial cells, as seen for cells ***a, b, c, d, e, g,*** for example, are lodged or encased in a matrix that still persists in ***f, g, i, k'*** and in which the capillary network is probably lodged.

2. The epithelial cells are of very variable sizes.

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3. Taking one of the cells, ***g'***, as an example, we see that it is constituted at the periphery by a part which appears denser than the rest of the cell, and which constitutes a framework formed very visibly by a branch ***k,*** coming from the interior of the inter-alveolar septum; this framework becomes much thinner at ***k'.*** We will see later that this is so because the framework is not located in a plane, but is convoluted in a spiral over 1 turn ½ to 2 ½; it is in the empty space constituted by this spiral that the cell is constituted.

For many cells, the framework is only seen on a portion of the periphery of the cell because of this spiral arrangement. Sometimes, it is completely detached from the cell which thus appears to lack it; often, it is found alone in the vicinity.

Pl. 14. *Section of tuberculous lung. Grosst 335. Agr. 600.*

4. From the frame come expansions which go towards the nucleus and which contribute to forming it as for the cells, ***m, g'***, for example; this fact can only be verified by the examination of a large number of cells; this examination can already be done on figure 1 of plate 15, but can be repeated so many times in the various figures of this work that I will not insist further here.

5. A peculiarity of the greatest interest, which we will encounter very often elsewhere, must be noted now. We see in cells ***g', m, n,*** plate 15 that the expansions or filaments, which leave the frame to move towards the center, actually start from a refracting or more or less colored ball forming part of this frame; we see these balls and the filaments they emit in the three points ***O*** of cell ***g';*** each filament, quite short, is terminated at the other end by another granulation, which gives it the appearance of a dumbbell (examine with a magnifying glass).

We will note this arrangement in other cells, notably ***l, g, h, i,*** figure 1 plate 15. Let us add further that, in the constitution of the cell, for any part and for each filament that we can observe in isolation, we will always see them constituted in the same way by dumbbell elements.

This is the fundamental principle of the cell's scaffolding. We will see later that the cell frames are also made up of a series of dumbbell-shaped rods located along their entire path, the latter being, by this fact, a bundle of dumbbells or fibrils composed of dumbbells placed end to end.

This constitution is not specific to the cell; it is that of all the elements that make up the inter-alveolar septum, various fibers or bundles that constitute what I call the carcass of the septum, vessel walls, etc.

This constitution can be seen precisely in most of the figures in this work, and we will return to it frequently. It can be seen with complete clarity in **Plate 18**.

Figure 1, **Plate 15** shows an alveolar wall cut obliquely at ***B*** and a range of epithelial cells in which the spiral-forming bundles or frames ***a, b, c, d, e, f*** are very clearly distinguished, as are the fine radiating branches which, starting from the frame, come to form the nucleus by their convergence; this arrangement is especially visible in the cells ***g, i.*** We can also observe, in many places, the existence of dumbbell-shaped rods which are straight or curved in a variable way up to the shape of a half-circumference.

We now know enough about the constitution of epithelial cells and the characteristic details of their conformation to be able to identify them with certainty and not to confuse them with lymphatic cells, for example.

Pl. 15. *Section of tuberculous lung. Fig. 1. Gross 335. Agr. 600. Fig. 2. Gross 1,100 Agr. 2,000. Fig. 3. Gross 600. Agr. 1,000.*

We will now examine the free cells in the alveolar cavities to determine their nature.

Figure 2 of **Plate 15** represents one of these very large free cells in a cavity. I have reproduced it here, greatly enlarged, to clearly show its identity with the epithelial cells and to complete the information we have already collected previously.

It is a cell already in the process of disorganization, but which nevertheless shows a very interesting peculiarity: the spiral formative framework ***b,*** which the section only began to attack in ***a,*** becomes much wider in ***c;*** it is impossible to say whether this is the point of origin of the formative bundle on the carcass of the inter-alveolar septum, or whether, on the contrary, it is the point where it spreads out into the cell to form it; I think that the latter explanation must be accepted. If we examine this framework under a magnifying glass in various regions, we see that it is made up of dumbbell-shaped elements.

This cell has two nuclei. It could be mistaken for an epithelioid cell or even, at a pinch, for a lymphatic cell. Its spiral-shaped framework formally indicates its epithelial origin.

Figure 3, **Plate 15**, contains a spiral cell frame ***a, b***, seen from the side, or, if you prefer, in profile; it is formed by a filament ***c*** which makes about two and a half turns of a spiral, and widens at the end of its path as shown in Figure 2.

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It is very rare for degenerated cells to present their frame in this way; fine filaments connect the turns of spirals together and these emit small branches which penetrate into neighboring elements.

Figures 1, 2, 3, 4, of **Plate 16** show the contents of various alveolar cavities; if the cells are examined successively, one or other of the characteristic arrangements of epithelial cells is noted; their type, shape, and size can be found in the epithelial cells of Plates 14 and 15.

As can be seen by referring to **Plate 14**, certain cells, much smaller than the others, are enclosed in the large tracts which form part of the supporting structure of the alveolus, such as ***a, b,*** in tract ***c*** (Fig. 1), ***d*** in tract ***e*** (Fig. 1), then ***a b, c*** (Fig. 4).

This fact is of great importance, which I must point out now; these cells, such as ***a, b, d,*** figure 1, are those which give rise to tracts such as ***c, e*** of the inter-alveolar septum; the embryonic cells of the tubercular tissue, which are about the same size as them, give rise to similar tracts which constitute the tubercular tissue, as can be seen in plates 32, 40, 41.

Pl. 16. *Desquamated epithelial cells. Gross 335. Agr. 600.*

This is not surprising since it has been demonstrated that the inter-alveolar septum (that is to say, the trabeculae such as ***c, e,*** figure 1, which constitute its carcass) forms embryonic cells by budding.

Another cell, of particular shape***, d*** (fig. 3, pl. 16) is formed by a spiral branch, its part ***e*** coming to engage under part ***d,*** much larger in part ***j,*** and emitting branches ***f, y, h,*** which connect it to neighboring elements. Outside this frame, the cell has only a nucleus enclosed in the cavity of the frame. It is probable that these very variable cell shapes are governed by their situation in relation to the network of blood capillaries and to the other epithelial cells.

The largest cells ***f*** (fig. 1, pl. 16), ***k, l,*** (fig. 3), ***a*** (fig. 4), are provided with a frame which clearly demonstrates their epithelial nature. By examining them under a magnifying glass, one will see the dumbbell-shaped elements which constitute them

In Figure 1 (pl. 16), cell ***g*** has a frame visible at ***h,*** which comes from a formative branch ***i*** that also contributes to the formation of other elements ***j*** and ***k.***

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In Figure 2, a large cell whose frame is visible at ***b, c, d,*** shows that the filaments emitted by the formative frame constitute, by their entanglement and anastomosis, a network, an areolar tissue that fills the space that, in the cell, is described as usually occupied by protoplasm. Examining this network under a magnifying glass, we see that it is formed by dumbbell-shaped elements of varying sizes, which appear in any case, face-on, obliquely, or end-on; in the latter case, and depending on their position, we see only one ball, or two balls placed side by side.

In summary, **Plate 16**, while increasing our knowledge of the constitution of the epithelial cells of the alveolar wall, also shows that all the cells observed in the alveolar cavities, in areas of tuberculous infiltration, are indeed epithelial cells.

The following plates will give us further details.

Plate 17 shows us an alveolar cavity ***A*** containing desquamated epithelial cells and a portion ***B, G, D, E, F,*** of the inter-alveolar septum carcass, cut somewhat obliquely. We will first note the beautiful demonstration of the mode of formation of epithelial cells, constituted by cell ***a*** and its formative branch ***b*** emanating from branch ***B*** of the alveolar septum carcass, to which it is still adherent; this cell ***a,*** with its formative pedicle ***b*** emanating from the septum carcass, clearly shows that, in reality, the embryonic cell whose pedicle is also born on the septum carcass is a young epithelial cell in the process of formation and that its physiological role is the restoration of the epithelium whose disorganized cells have fallen into the alveolar cavity.

Pl. 17. *Desquamated epithelial cells. Gross 550. Agr. 1,500.*

Other cells complete this demonstration, for example: cell ***c*** connected to the septum by two short branches, one of which is broken, and whose frame ***c*** emitted a branch ***d*** contributing to the formation of a neighboring epithelial cell; cell ***e*** with its branch ***f,*** also detached; cell ***i*** with its branch disintegrated into two parts ***g*** and ***h.***

We also have here the demonstration of the constitution of the bundles or large fibers or tracts, which constitute, by their anastomosis, the carcass of the inter-alveolar septum. We note, in fact, that the bundles or tracts ***F, B, G, C, D, E,*** are made up of dumbbell-shaped elements. All the cellular elements, as well as all the bundles or filaments visible in the figure, have an identical constitution.

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We see, everywhere, only dumbbell-shaped elements differing from each other either by the size of the balls or by the length of the filament which connects them. We will become aware of this constitution by exploring, with a magnifying glass, the tracts of the inter-alveolar septum ***B, C, D, E, F,*** as well as the regions ***h, n, l, p.*** We will see, by the balls placed on its path, the constitution, by dumbbells, of the filament ***h***; in ***j,*** the ball ***k*** has formed a dumbbell whose rod is quite long and of which we can barely distinguish the other ball. The large granulation ***o*** forms a long and large dumbbell-shaped rod which constitutes the proof of the fact that the residual elements of the desquamated epithelial cells vegetate and multiply. We will see as much at multiple points in the figure. Cell ***l,*** for example, gives birth on its left to a dumbbell-shaped element whose external ball has formed an embryonic cell which detaches about a centimeter outside the cell.

Three other strongly stained embryonic cells, one above and to the left of the figure, the other in the center, the third at the bottom and in the middle, have an identical mode of formation very visible in the one at the top.

The large free cells in the alveolar cavity also show a framework and its composition. Empty of its contents, a cellular framework is seen at ***p,*** showing the granulations of the dumbbells that constitute it.

In cell ***a,*** these are dumbbells which, starting from the frame, converge towards the central nucleus.

One could object to the arrangements I describe, that they do not represent the normal state of the anatomical elements, since they are elements in the process of decay or disorganization.

The objection would be unfounded because, whatever this state of decay, at the beginning or already advanced, the fundamental constitution of the cells always reveals itself to be the

same, as it is, for example, in cell ***a,*** plate 17, whose state of decay is so little advanced that it is still attached to the inter-alveolar septum by its pedicle or formative branch.

A fine demonstration of this cell constitution is given in plate 8 by cell ***C,*** still included in the inter-alveolar partition and which shows its nucleus, the radiating dumbbells which connect it to the frame, then at ***R*** and at other points the branches which, starting from the inter-alveolar partition ***B D,*** form this frame and the cell itself.

The arrangements that I describe are therefore indeed those of normal elements; but never-the-less, they are better seen and are easier to observe in elements in the process of decay, because the isolation of these of the wall and above all, probably the loss of the albuminous

liquids which they contained leaves them, after the action of the coloring reagents, a transparency making visible the details which would not be perceptible under other conditions.

Plate 18 gives further confirmation of this important fact: the formation of epithelial cells by branches emanating from the inter-alveolar septum.

In this figure, we see the traces of the inter-alveolar septa ***d, f, g, h, k,*** in the process of dislocation. The alveolar cavities contain both cell and alveolar wall debris and embryonic cells. In ***a,*** we see a cell, also in the process of dislocation, of which only a part of the internal framework ***a*** remains, but which has remained adherent to the alveolar wall by two or three formative filaments ***c, i, b,*** which we see leaving the septa, either at ***j,*** or on the portion of the septa ***e,*** detached from its other part ***d.*** Thus, this cell is indeed made up of branches emanating from the inter-alveolar septa.

Let us make a few other observations in this figure that reinforce the previous conclusions. At ***l,*** we see an epithelial cell still adherent to the dislocated wall ***d.*** This cell is composed of a frame ***m,*** from which radiating dumbbells depart and converge towards the nucleus ***n.***

Next to it, at ***o,*** we see a small embryonic cell whose club-shaped pedicle is a very clearly visible dumbbell ***p.***

At ***q*** and ***r,*** two embryonic cells are formed, born on the branches ***i*** and ***b*** that form the epithelial cell ***a;*** two embryonic cells, almost sessile, are born side by side at ***t*** on a filament; we see other typical embryonic cells with their pedicle at ***u, v, x, y***..., etc., and at the places marked with a cross.

In **Plate 19,** we also have confirmation of the previous observations.

In ***A, B, C,*** we see an inter-alveolar septum cut very obliquely, almost in its plane in certain places, then other septa ***D, E, F, G, H***, cut transversely and partly dislocated.

Pl. 18. *Section of tuberculous lung. Gross 335. Agr. 600.*
Pl. 19, *Section of tuberculous lung. Gross 335. Agr. 600.*

We will first observe that, in the carcass of the septum, ***B, K, L, G,*** epithelial cells such as ***a, b,*** have remained in place.

In the two alveolar cavities ***M, N*** (the alveolar septum ***G*** which separates them is dislocated), there are free cells, in the process of destruction, detached from the wall and in which we recognize the formative elements, framework, radiating filaments and nucleus.

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Finally, we will especially note the arrangement that this plate has for purpose to show, the formation of epithelial cells by branches emanating from the wall. We will see three of these cells ***c, d, e***, and ***f,*** still joined to the wall by one or two formative branches. If we examine the reticular tissue of the cells in the process of dislocation at points ***g, h, i, j, k,*** we will see numerous dumbbells forming embryonic cells.

Let us now recapitulate the different facts that result from the examination of **plates 15 to 19.**

1. The inter-alveolar septum comprises a carcass forming a framework which serves both to support the various constituent elements of the wall and to form them.

2. Epithelial cells are formed by one or more branches originating from the supporting carcass of the septum and which form, around the cell, a framework rich in chromatic substance connected to the nucleus by radiating filaments consisting of dumbbell-shaped elements. The constituent branches of the cell are made up of dumbbell-shaped elements placed end to end and side by side to form a bundle.

3. The radiating filaments, emanating from the formative bundles, anastomose with others to form a reticular tissue consisting of dumbbell-shaped elements and which occupies the space of the cell currently indicated as occupied by protoplasm.

4. The free cells in the alveolar cavities in regions of the lung invaded by tuberculous infiltration are desquamated epithelial cells, as many observers have affirmed, and they have nothing in common with lymphatic cells.

5. The figures show that the desquamated epithelial cells degenerate and become disorganized. Because of this disorganization, they do not evolve by fusing with others to transform into giant cells.

6. The elements remaining after the disorganization of the inter-alveolar septum-tracts, desquamated cells, formative branches, filaments, and granulations-remain alive; it is only the organization of the cell that is lost, and these elements can give rise to other granulations and embryonic cells capable of emitting the filaments that constitute the tuberculous tissue, as will be seen later.

We must retain, from these facts, those that relate to the formation and general constitution of the cell. These facts obviously have a scope that is not localized to the epithelial cells of the pulmonary alveolus. Some of them must apply to the constitution of cellular elements in general.

Every cell is constituted by a formative branch whose elements originate from a distant point. This branch emits branches that constitute the cell by their ramification and anastomosis; the branch, as well as the entire cell, are constituted by dumbbell-shaped elements which are, in reality, those that have been called mitochondria and of which neither the real form nor the role are currently known.

I had already explained this mode of cell formation in the initial studies that are the subject of the first volume of this work.

It has been described **(1)**, for example, regarding the constitution of liver cells; but what I had not seen or explored sufficiently was the elementary constitution of the cell's formative branch and the elements that constitute its areolar tissue and the nucleus itself. This constitution, visible in several of the preceding plates, opens the way for us in the study of the organization of living matter.

This constitution of the cell's formative branch and of the entire cell by dumbbell-shaped elements is, in reality, the fundamental principle of the organization of the living being and of life. The dumbbell-shaped mitochondrion is the constructive element of the organization and of the entire organism, whether animal or plant.

These dumbbells will form the solid felting that constitutes the membranes, vessels, nerves, cells, that is, the entire organization of the individual. Whether the granulation of living matter, chromatic, nuclear, remains in the organism or is transported outside, in an in vitro culture, the property of emitting its dumbbell-shaped filament remains intact; only the faculty of organization varies.

We will see in **Plate 19** that the chromatic granules of embryonic cells, rejected with the caseous matter resulting from the destruction of the lung tissue, have retained their property of germinating and that they give rise to dumbbell-shaped filaments that are sometimes quite long.

As these filaments give rise to new granulations which in turn form new ones, we see that this ensures the continuous renovation or rejuvenation of matter and anatomical elements; at the same time, in growing organisms, the lengthening and increase in the

(1) *"Constitution of Animal and Plant Organisms" p. 133 and Plates 89, 90, 92.*

dimensions and volume of anatomical elements and organs are ensured by this same process. This is probably how the regeneration of a severed nerve can take place.

It seems that the dumbbell-shaped filaments or knotted ropes must be able to constitute an uninterrupted bundle of a very great length, several meters if you will. The constitution of such a bundle is achieved in the myelinated nerve fibers, which are entirely, and since their birth, a continuous bundle of mitochondria. This fact is developed in a following chapter.

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STUDY OF THE GIANT CELL

According to **Yersin**, then **Borel** and other authors, the giant cell results from the fusion of a certain number of large mononuclear leukocytes *which come together around the bacillary clusters to encompass and destroy them;* the protoplasmic expansions with which they are provided would be directed towards the bacillary clusters for this purpose of destruction.

The origin of giant cells is still highly debated today.

For some authors, **Dominici** and others, they arise from the coalescence of epithelioid cells, these in turn coming from the fixed cells of connective tissue, or from lymphatic cells.

Here is how **Borrel (1)** describes the formation of giant cells:

''*With **Krause, Metchnikoff,** and many others, I believe that the giant cell results from a process of pure and simple conglomeration*.''

''*The injection of bacilli into the circulatory system shows us this mode of formation with the greatest evidence. From the third day, the sections show numerous giant cells inside the vessels themselves, and we see them forming, so to speak, around the bacillary clusters.*''

''*Here, their origin is not in doubt; it is through the fusion of mononuclear leukocytes that they arise. The number of nuclei in such protoplasmic masses can sometimes be very considerable; I have been able to count up to sixty. Very often, in the same plasmic mass, the nuclei are arranged in groups and almost always in a collar at the periphery. The arrangement of the nuclei at the periphery does not seem very difficult to understand if we take into account the fact that, in any mobile cell, it is always the part deprived of a nucleus that progresses, the portion of the cell containing the nucleus is always the lagging part. Given a bacillary mass, we see the mononuclear leukocytes, located on the vascular wall,*

(1) *Borrel, loc. Cit.*

sending out expansions in the direction of the bacilli, the nucleus always remaining at the periphery, these pseudopodia are sometimes very long and the giant cell results from the progressive confluence of a large number of these extensions.

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In some cases, all the nuclei are concentrated at one pole and the bacilli are located in the part of the cell deprived of nuclei. This localization of the bacilli in the portions deprived of nuclei has been noted by all observers. It seems to me to be explained very simply by the hypothesis I put forward above.''

Kostenich and **Volkow (1)** have disputed that giant cells emit expansions or pseudopodia. They claim that *"Under the action of fixing liquids, the giant cell retracts, detaches itself from the surrounding elements and the parts having penetrated into the intervals of the latter take the form of branched appendages. The modified reticular fibers and those of the connective tissue, by fusing with the giant cell, contribute to the formation of these appendages.*"

According to **Maximov (2)**, the giant cell would result from a plasmodium constituted by a group of epithelioid cells. These would themselves be formed either by lymphoid cells or by cells of the reticuloendothelial apparatus.

According to **Hollande (3)**, "*the giant cell is only a simple hypertrophied epithelioid cell whose asteroid cavity, increasing considerably in volume, takes on enormous proportions and ends up occupying the entire cell, rejecting at its edges the protoplasm which remains slightly basophilic.*"

''The numerous nuclei that are characteristic of the giant cell are found only in the protoplasm; they can therefore only be located at the periphery of the cell. All originate from the single nucleus of the primitive epithelioid cell. Nuclear multiplication is established here by amitotic divisions of the primitive nucleus and the newly formed nuclei derived from it, and not by karyokynesis.''

Verson and **Fananas** noted the *presence of the Golgi apparatus* in epithelioid cells and in giant cells; it occupies the central part of the giant cell and clearly presents a reticular appearance; **A.-Ch**. and Mrs. **G. Hollande,** who sought to demonstrate it, never succeeded in observing a network of filaments in giant cells.

(1) *Kostenich and Volkow, loc. cit.*
(2) *Maximow, Tuberculosis of Mammalian Tissues in Culture, Ann. d'anat. Nath. med. Chir. - No. 1, January 1926.*
(3) *Hollande, loc. cit.*

I have observed, either in the center or at the periphery of the tubercular follicles of the human lung, or in infiltration masses, the most variable forms of giant cells; we will study them, but we will first examine several giant cells of typical form to show their constituent elements.

Let us take as a first example the giant cell located in the center of **plate 20**; this cell is represented, enlarged, in **plate 20bis.** We observe there:

1. Throughout the interior a distinctly areolar tissue ***a.***
2. In the center, a mass ***b*** a little more colored.

3. At the pole opposite the one containing the most nuclei, six or seven protoplasmic expansions, ***f, f, f,*** very long, which ramify, and whose branches anastomose with neighboring branches. These anastomoses are not artificial creations due to preparation maneuvers or chemical reagents, *because they are constituted, even in their ramifications, by the same areolar tissue which constitutes the entire interior of the giant cell.*

On these expansions, or on their branches, embryonic cells ***g, h, i, j, k, l, n, o, p, q*** are born, in various states of development and of different sizes; finally other embryonic cells such as ***m, n, v, r,*** are born on the expansions of the giant cell and immediately towards their point of exit.

If we examine all the embryonic cells contained in **Plate 20**, we quickly notice that they are identical to the nuclei of the giant cell. It should also be noted that some of these embryonic cells ***x, x, x***, are equipped with a very distinct club-shaped pedicle, which is a dumbbell-shaped element of which we only see the rod and the external ball, the other ball being in the embryonic cell and its origin.

We cannot follow the expansions very far, because they themselves, or their branches, will anastomose and get lost in other branches not far from the cell. The fact that these branches give rise to embryonic cells, as we will see later, and that they have the same areolar structure as the contents of the cell, is sufficient to to identify and prevent them from being confused with artificial creations of another nature.

The intermediate framework between giant cells and others is constituted exclusively by fibrillar and areolar tubercular tissue, and the expansions of the giant cells contribute to forming this framework in which their ramifications are lost.

Pl. 20. *Giant Cell. Gross 335. Agr. 600.*
Pl. 20 bis. *Giant cell. Gross[1] 770. Agr. 1,400.*
Pl. 21. *Giant cell. Gross 1,100. Agr. 2,000.*
Pl. 22. *Giant cell. Gross 1,100. Agr. 2,000.*

4. At one pole of the cell, about ten nuclei ***c, c, c,*** laterally one or two nuclei ***d*** on each side, and at the other pole, five or six elongated nuclei ***e, e.***

We will examine successively the particularities that I have just noted in the giant cell.

1. Interior content of the giant cell.

It is always constituted entirely by tissue with a very clearly areolar, reticulated appearance.

The shape of this tissue is represented in plate 20 bis and in plates 21 and 22.

The areolar framework is constant; it is a fundamental characteristic of the giant cell, as well as of epithelioid cells; this areolar structure is also constant in the more colorful central part.

Plate 21 shows a giant cell in very thin section, a little torn, at a magnification of about 2,000. We can see at the periphery a certain number of nuclei, ***a, b, c, d, e, f, g, h, i, j, k, l, m***.

We see in ***n, o, p, q, r, s, t,*** expansions of the cell; in ***A,*** an oval part that is a little more colorful, like a large central nucleus. As for the areolar tissue ***B,*** we see it formed by dumbbell-shaped elements. The granulations of the dumbbell-shaped elements are of very different sizes, some very small, others two or three times larger.

Plate 22 represents another giant cell, at a magnification of approximately 2,000. The same observations can be made on this cell that were just made on the previous giant cell. I have reproduced these two cells at such a high magnification because they constitute a precise control and even research document that allows the reader to study for himself, as well as, and even better than, my original preparation, the constitution of giant cells.

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In addition to the constituent parts already mentioned above in giant cells, the following facts can be observed in this one:

A) The nuclei a, b, c, of the giant cell contain large granules.

B) These granulations emit fine filaments that can be seen emerging from the periphery of the nuclei and which themselves carry another granulation at their end; these are dumbbell-shaped rods. They can be seen all around nuclei ***a*** and ***b;*** the first nucleus ***c,*** to the left of ***b,*** is pyriform and shows its very clear, isolated formative pedicle, which is a dumbbell-shaped rod carrying, also very clearly at its end, its free ball, the other being inside the nucleus. This image demonstrates in an indisputable, irrefutable way that the

peripheral nuclei of the giant cells are embryonic cells identical to those observed everywhere else in the tuberculous tissue.

The reader will be able to see numerous other dumbbell rods in all other parts of the figure and note that it is their varied arrangement that constitutes the areolar tissue of the giant cell, including the central part; this areolar tissue appears to be constituted exclusively by the dumbbell rods and their granules; these are of variable coloration, the most colorful being those rich in chromatic matter; the least colorful have lost it.

The shape of the areolar tissue of the giant cell, as well as the constancy of its existence, can be verified in figures 3 and 4 of plate 24 and in plates 25, 26, 27.

C) By examining the embryonic cells contained in the figure outside the giant cell, we see that they have a constitution identical to that of the nuclei of the latter. They contain granules of different sizes, according to the degree of their development.

The granules are small in young cells and large in older cells; they have the same dimensions as those of the nuclei of giant cells, for example, in cells ***d, e, f, g.*** In ***h,*** a young elongated embryonic cell is seen in which the granules in the process of formation are clearly distinguished. In ***k*** and ***l,*** two adult embryonic cells are seen, whose granules are ready to escape, and which will be formed outside, but immediately beside, the giant cell; their identity with the nuclei ***i*** and ***j,*** for example, of the latter is noted, a fact which adds to others already explained to demonstrate that the nuclei of giant cells are embryonic cells identical to the others.

2. Constitution of the central zone of the giant cell.

In the middle of the giant cell, that is, in the middle of the mass of areolar tissue, we notice a slightly more strongly colored zone. Examination of this zone shows that its constitution is identical to that of the areolar tissue, without any difference.

3. Constitution of the expansions of the giant cells.

These expansions, denied by some, are considered by others either as artificial formations or as formations analogous to the pseudopodia of leukocytes.

It is enough to examine **Plate 20 bis** to be convinced that these expansions do indeed exist and are constituted by areolar tissue identical to that of the giant cell. They are the continuation, on the outside, of the formation of this areolar tissue, this areolar form being the one that tuberculous tissue takes everywhere.

In the following plates, we will find other examples that demonstrate, beyond any doubt, this constitution of the expansions, their origin, and their role.

They are formed exclusively, like areolar tissue, by dumbbell-shaped elements. We have observed so far:

1. The existence, elementary constitution, and arrangement of the areolar tissue of the giant cell.

2. The origin of this tissue formed by dumbbell-shaped elements born from the granulations of the nuclei of giant cells.

We will now give, from this origin, new demonstrations that lead us to determine the nature and origin of the peripheral nuclei of giant cells.

Nature, origin, and evolution of the peripheral nuclei of giant cells.

Plate 23 provides the demonstration we are looking for; this plate represents a region of the lung invaded by infiltrating tissue that is destroyed in ***A,*** where we see the emptiness of a small cavern.

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In ***B,*** there is a giant cell whose formation is not yet very advanced because its areolar tissue is only at the beginning of its formation; it is to this fact that we owe our very clear observation in this giant cell of the germination of nuclei.

The giant cell ***B*** has two groups of nuclei at the two poles ***C*** and ***D.*** Each of these nuclei emits one or more dumbbell-shaped filaments that run toward the center of the cell. One of the nuclei ***a*** emits dumbbell-shaped rods all around it, at ***b,*** at ***e,*** and on both sides. Here, all the nuclei are visibly in a state of germination.

Note the important fact that certain nuclei, as in regions ***d*** and ***e,*** not only emit their germinative filaments toward the center of the cell but also emit them in the opposite direction and thus constitute the constituent material of the outer expansions of the giant cell, as in ***e.***

These germinative filaments emitted by the nuclei of giant cells and by embryonic cells are those that different observers, notably **Borrel,** have considered to be pseudopodia emitted by the mononuclear leukocytes that form the giant cell to destroy Koch's bacilli "always located in the center of the cell."

Let us say from now on that these germinative filaments or pseudopodia, consisting exclusively of dumbbell-shaped elements, are Koch's bacillus itself, formed by the germination of embryonic cells.

Pl. 23. *Giant cell. Gross 335. Agr. 600.*

Thus, quite obviously, the dumbbell-shaped filaments or simply the dumbbells formed by a nucleus of a giant cell, come from the germination of the granules contained in this nucleus. Let us add here that other very complete demonstrations will be given later of this fact and will confirm it.

Let us now examine the other points of the figure. We see, on all sides, nuclei identical to the peripheral nuclei of the giant cell ***B*** and which are embryonic cells; to complete the identity, a large number of these cells emit germinative filaments like the nuclei of the giant cell.

Here we come to a difficulty that must be noted immediately. We know that the embryonic cell is born at the end of a pedicle, often club-shaped, itself born from another filament or a bundle; we have seen that this club is a dumbbell-shaped rod with a ball hidden in the embryonic cell.

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It happens that at a given moment, when the embryonic cell has gone through its normal development and continues it by the germination of its granules and the emission of germinative filaments, it may be difficult to recognize whether, for certain cells, the filament emitted is the formative filament or the germinative one. This difficulty only exists if there is only one germinative filament; when the cell enters into germination, it emits germinative filaments at different points around its circumfe- rence, which emanate from different chromatic granules. This germination of cells will be explained later with all the necessary demonstrations.

The embryonic cell that has not yet germinated is strongly basophilic and stains violently with Ziehl fuchsin; it is acid-fast; the embryonic cell, whose germinative filaments have already been emitted, only stains weakly with Ziehl. However, at the beginning of emission, it still stains strongly.

A. Ch. and Mrs. **G. Hollande** have already clearly seen that embryonic cells are less and less basophilic as they age.

With the help of these characteristics, we will see:

1. In ***f, g, h, i, j, k, l, m, n, x,*** **plate 23,** embryonic cells having emitted their germinative filaments, some being, as a result, in the process of disappearing, their role having been fulfilled.

2. In ***s,*** for example, an embryonic cell equipped with a club-shaped pedicle which is a dumbbell, and which emits two or three germinative filaments at its periphery. In ***u,*** we will see the dumbbells forming two embryonic cells with their free terminal ball.

The nuclei of the giant cell which have already emitted their formative filament, but whose emission is not complete, still stain notably with Ziehl fuchsin, but however already very clearly less than the cells which have not yet begun their germination.

I recall here that we have already seen in **plate 8** the germination of the embryonic cells of the group of cells ***L, M, N;*** we will see in ***k,*** in this plate 8, the very clear dumbbell-shaped filaments resulting from this germination.

Plate 24 gives another example of two young giant cells, ***C (***Fig. 1) and ***C*** (Fig. 2), whose nuclei are in the process of germinating to form areolar or reticular tissue. In cell ***C*** (Fig. 1), we see that all the peripheral nuclei emit their dumbbell-shaped filaments.

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In summary, the facts we have just presented demonstrate:

1. That the nuclei of giant cells are embryonic cells identical to those existing in the neighboring regions of these cells.

2. That the areolar tissue of the giant cell is formed by dumbbell-shaped germinative filaments arising from the granulations of the peripheral nuclei.

3. That the peripheral nuclei have an evolution identical to that of embryonic cells and which results, in both cases, in the formation of reticular and areolar tubercular tissue.

4. That the expansions of giant cells are the continuation, on the outside, of the formation of areolar tissue.

Thus, the constitution of giant cells is established by these facts, a knowledge which entails that of their origin, since they are formed by a group of embryonic cells whose origin has been previously established.

Let us emphasize how these material facts, which result exclusively from direct observations by photography, and in which no hypothesis intervenes, agree with the fact that embryonic cells are formed on the spot and are not diapedesized lymphatic cells.

Let us add, to complete the demonstration, that we will see in the figures which follow, that the nuclei of giant cells can be born on a peripheral branch entering into the constitution of the cell and that this same branch often emits, outside the latter, ordinary embryonic cells; it results, by the fact of this common origin, the identity of their nature and their constitution.

Moreover, an identical demonstration has already been given in the preceding pages by the fact that the protoplasmic expansions of giant cells, which are formed by the areolar

Pl. 24. *Giant cells. Fig. 1 and 2. Gross 335. Agr. 600. Fig. 3 and 4. Gross 550. Agr. 1.000.*

tissue coming from the peripheral nuclei, give rise to normal embryonic cells, outside and far from the giant cells.

The facts we have just presented completely enlighten us on the significance and role of the giant cell. This significance is null; the giant cell is in no way the typical tubercular element; its role is exactly the same as that of the other elements of this tissue, and this accords perfectly with the facts that **Grancher** was one of the first to present and which led him to conclude that the form of the tubercular tissue is of no importance.

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In summary, a giant cell is made up of a group of embryonic cells which, by germination, form the areolar tissue of the cell body, a tissue which continues to vegetate and extend outward in the form of expansions forming part of the intermediate matrix or gangue.

We will see later, and after the study of the tuberculous infiltrating tissue and the framework that constitutes it, that the giant cell simply corresponds to a form of this tissue in which the embryonic cells, which everywhere are the agent of formation of the tuberculous tissue, are grouped in a complete half-crown, instead of being scattered and without any order as they are in the infiltrating tissue. It is the latter and not the giant cell that represents the characteristic form of the tuberculous tissue.

Our knowledge is now almost complete on giant cells. We have only a few particular points of secondary importance to examine; during this examination, we will have the opportunity to study some other types of giant cells and to make new observations that will confirm and complete the previous ones.

We will study the following points successively:

1. The cause of the formation of giant cells and their particular arrangement.
2. Some diverse forms of giant cells
3. The place of formation of giant cells. Can a giant cell be the formative center of a tuber?

Causes of the formation of giant cells.

The main cause of this formation appears to be the chance arrangement occupied by the embryonic cells at the moment they enter germination.

Giant cells have the shape we observe because they can only form *when embryonic cells are arranged at the periphery of an empty space.*

It will be easy to see by quickly scanning a number of plates in this volume that embryonic cells are born without any order. We will see in certain plates, notably plate 4, that

they are often born in clusters on the same formative filament, that is to say in groups, or in a double row along a straight or curved filament.

As a result, it happens that a group of embryonic cells is arranged in a half-crown or even a crown **(1)**, around a space not occupied by other cells. In this case, the germinative filaments of these cells will head into the free space, that is to say towards the interior and will thus constitute a giant cell.

It is obvious that giant cells cannot form if this arrangement does not exist. If the embryonic cells are arranged in a tight cluster, they germinate toward the outside of this cluster. If they are spaced and scattered, they germinate in all directions.

There are, moreover, giant cells in which the nuclei are not all peripheral and are disseminated from one pole to the vicinity of the center: witness the cells in Figures 1, 2, 5, 7, **Plate 17.**

The arrangement that can most easily give rise to a giant cell is that of a large cluster of 30 to 50 or more embryonic cells, in the center of which there is a void.

As for the formation of giant cells by fusion of several epithelial cells, the study we have made of the latter clearly demonstrates the impossibility of such an evolution; on the other hand, the demonstration of the nature of the peripheral nuclei of giant cells and of the origin of embryonic cells constitutes at the same time the demonstration of the origin of giant cells.

Various forms of giant cells.

The most remarkable of these forms is that of the spherical giant cell represented in plates 25 and 26; this cell is formed by a complete crown of peripheral cells. Like the preceding ones, it is formed by a mass of areolar tissue b more strongly colored in the center a than at the periphery.

At two different points ***A*** and ***D,*** two branches or groups of branches can be distinguished at the periphery of the cell, neither of which has the appearance of expansions which, as we saw above, are formed by areolar tissue. Here the coloration of these branches, much darker than that of the areolar tissue, indicates that they are not an expansion of the latter. On the other hand, the group of branches ***A*** (we can distinguish at least two ***c, d***), divides into a series of others which appear to form a lascis at the periphery of the cell.

(1) *It would be more accurate to say arranged in the form of a cap, or arranged over the entire surface of a sphere, because the giant cell is not flat, but globular.*

We see, for example, the clear beginning of two branches, one at ***G,*** another at ***K,*** **plate 25** and others which head into the region ***l,*** m, from where we see the pedicles of several cells which, as we saw above, are embryonic cells; we can doubt this even less since we see several being born, outside the cell on this group of branches ***A;*** we see one ***g*** with its very clear pedicle at ***A,*** another ***f*** with its pedicle ***e;*** there is one at ***p*** arising on the branch and one next to it ***p'*** arising on the branch ***w*** (**plate 25**).

In ***D*** and ***E,*** there also appears to be a group of branches that also send out branches ***L, w, r, s,*** (Fig. 1, **plate 26**) spreading out to the periphery of the cell. These branches cannot be taken for expansions formed by the cell, precisely because of this spread-out arrangement at the periphery of the latter because, upon leaving it, the expansions move away from it in an eccentric direction which is roughly that of a ray starting from the center.

Figure 1, **plate 26**, is a photograph of the same cell, but with a different focus.

The ***L*** branch alone provides a fine demonstration of the origin of the peripheral nuclei and their quality as embryonic cells. Indeed, we see this branch emitting in Figure 1 of **Plate 26**, the small branches all marked with a line and a cross which form peripheral nuclei. We can clearly see some of them at the end of these short branches. The ***L*** branch therefore forms a cluster of very tightly packed embryonic cells; this well justifies the explanations I gave above on the causes of the shape of giant cells, **page 68.**

In the ***H K*** region, Figure 1, **Plate 26,** the peripheral nuclei also constitute a cluster in which the pedicle of some of them is visible.

Some branches coming from the outside, such as ***F,*** also appear to contribute through their branches ***F' F'***, to the formation of the peripheral lascis and embryonic cells are seen forming on the branches of this lascis.

Let us add to this description that the group of branches ***A*** is the continuation of bundle ***B*** (pl. 25). We see, in fact, in Figure 1, **Plate 26,** that these two parts join on a common trunk ***C;*** this part ***B C*** appears to be a remnant of the carcass of an inter-alveolar septum. The same appears to be true of region ***E.***

In this case, the branches forming the clusters of nuclei and the peripheral lascis would, in reality, be emitted by an inter-alveolar septum. This corresponds perfectly to what we have already observed concerning the vegetation of the inter-alveolar septum and the formation of embryonic cells.

Pl. 25. *Giant cell. Gross 550. Agr. 1,000.*
Pl. 26*. Giant cells. Fig. 1, 2, 3. Gross 550. Agr. 1,000.*

Figures 2, plate 22, 1 to 4, plate 24, 2 and 3 of **plate 26**, and **plate 27** contain a series of photographs showing various forms of giant cells; the presence of the elements just described can be observed there, and the facts set out in this chapter can be verified under a magnifying glass.

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FORMS AND EVOLUTION OF TUBERCULAR TISSUE
INTERMEDIARY FRAME

Tuberculous tissue is the whole formed by this intermediate framework and by embryonic, epithelioid, and giant cells. It has been previously demonstrated:

1. That embryonic cells are born on filaments or branches originating either from the interalveolar septum or from other embryonic cells.

2. That embryonic cells germinate and give rise to dumbbell-shaped elements that can be arranged end to end to form fibrils.

3. That the expansions of giant cells are constituted by the vegetation, outside these cells, of the reticular and areolar tissue formed inside them by the vegetation of their peripheral nuclei, these being, in reality, embryonic cells.

Like the peripheral nuclei within giant cells, embryonic cells germinate wherever they are found and give rise to isolated fibrils or bundles that anastomose with others to form the intermediate web. In reality, it is not an intermediate web, since it is formed by the embryonic and giant cells from which it emanates.

Also, this designation of intermediate web is inaccurate, because what it represents is the entire tubercular tissue; none of its elements can be separated from the others, all *resulting from the evolution of a single one, the embryonic cell.*

It is therefore not only the intermediate web or gangue, but the tubercular tissue itself that we are going to study.

Many forms of this tissue can already be observed in the previous plates. Here are some new ones. One of the most common is that shown in **plate 28**; it is both the common form of tuberculous tissue in the tubercles and in the infiltration tissue.

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In ***A*** we see a zone of almost compact tissue, which no longer stains with difficulty with Ziehl's fuchsin and which is an area evolving towards caseification; then in ***C, C,*** a zone very rich in embryonic cells born on well-colored and very visible filaments, and finally an intermediate zone ***B, B***, in which embryonic cells are still seen, but which are, for the

Pl. 27. *Giant cells. Fig. 1, 3, 5. Gross 550. Agr. 1,000. Fig. 2, 5, 6, 7. Gross 335. Agr. 6,000.*

most part, almost completely discolored, ***s, t, u, v, x,*** because they have germinated or are in the process of germination, their chromatic granules giving rise to dumbbell-shaped fibrils.

These join together in twos to four or five elements or more, in bundles or trabeculae which anastomose and form large areoles such as ***fff;*** as the tissue ages, that is to say as the multiplication of the fibrils continues, these areoles decrease in size, then finally disappear, becoming compact tissue similar to ***A,*** this representing the oldest region and region ***C*** being the youngest. The tubercle therefore grows from its periphery, the most recently formed embryonic cells, that is to say the youngest, always being the furthest from the center.

Note that when examining region ***C,*** we will see many embryonic cells equipped with their formative pedicle; only a few are indicated by the letters ***i, j, k, l, m, n, o, p, q, r.***

Plate 29 represents, at a high magnification, a small region of the young tubercle from plate 1. We can distinguish the three zones that I have just indicated: zone ***A*** evolving towards caseification, region ***C*** of embryonic cells in the process of formation, and intermediate region ***B*** of embryonic cells beginning to germinate.

In zone ***G,*** we see that the embryonic cells ***r, s, t, u,*** for example, are in a state of maturation, staining very strongly with Ziehl fuchsin; one of them ***t*** is full of large granules.

In region ***B,*** the embryonic cells, which have begun to germinate, are only slightly stained; this is very visible on cells ***a, b, c, d, e, n,*** for example.

In region ***A,*** the embryonic cells are almost completely stained, and if only their silhouette can be distinguished, it is because of this absence of staining.

To inform the reader on this point, the outline of the embryonic cells has been marked with a dotted line in the region; in region ***v,*** where these silhouettes are also distinguishable, the center of a certain number of cells has been marked with a cross; in ***i*** and ***h,*** we will see several cells whose large granules are particularly clearly distinguished.

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Finally, it will be easily seen with the naked eye, and even better with a magnifying glass, that the entire region ***A,*** the center of the tubercle evolving toward caseification, *is in the process of becoming a mass composed solely of dumbbell-shaped elements and granules between which filaments run.*

In this **plate 29**, only the central part has been in focus for photography. But in the peripheral part, we can nevertheless see a great many isolated dumbbell-shaped elements, which were in focus.

Pl. 28*. Section of tuberculous lung. Gross 335. Agr. 600.*

Examination of these peripheral regions will clearly show the elementary constitution of the trabeculae of tuberculous tissue, and the arrangement of the dumbbell elements to constitute them. In ***D, E, F, G, H, I, J***, and ***K,*** we will see dumbbell elements arranged in parallel. Sometimes there are only two, other times three, four, as in ***E,*** or more.

In the ***M*** and ***P*** regions, we will notice that the dumbbell elements placed end to end constitute fairly long fibrils, arranged parallel in pairs.

In the region of the areolae ***x, x, x,*** etc., the dumbbell elements have been marked with small crosses. There, we can check the constitution of the trabeculae just indicated and the characteristics of the dumbbell elements.

There, we will see the formation of a large span.

We must now return a second time to the explanation of the contents of Plate 1. This figure is a photograph at a low magnification of the region of which **Plate 29** represents only the zone ***a, a.*** In the center, we see a mass of tissue ***a, b, b, b,*** in the process of evolving towards the caseous state; in ***c,*** a region of fibrous appearance constituted by bundles of anastomosing fibrils, the elementary constitution of which we have just indicated above, among which some seem to start from the central mass, while others start from the giant cells ***d, e;*** in ***f,*** we see a region where embryonic cells abound; in ***g,*** an alveolar cavity containing a few desquamated cells, scattered and dissociated desquamated cells ***j;*** in ***h,*** a zone where a large number of young embryonic cells are in the process of formation.

It should be noted, in this figure:

1. That the mass ***a, bbb,*** which is obviously a tubercle in the process of forming, is not round, but triangular in shape.

2. That the two giant cells, ***d, e,*** are at the periphery of this mass which, in its interior, does not contain any.

3. That the distribution of highly developed embryonic cells on one side, ***f,*** and not on the other, ***c,*** shows that there is no regular and circular development of the mass ***a, b, b, b*** here.

4. That the evolution towards caseification is advanced on one side only, region ***i,*** while region ***f*** is still only at the stage of embryonic cell formation.

5. That finally, the embryonic cells are very clearly at the stage of germination.

If we compare these observations with those already made regarding **plate 29,** which represents the enlarged region ***a,*** we are led to the following conclusions:

Pl. 29. *Section of tuberculous lung. Gross 335. Agr. 600.*

1. The mass ***a, b, b, b,*** which is, in reality, a tubercle in the process of formation, develops in a mass of infiltration tissue.

2. The cause of the formation of this mass evolving towards the caseous state is solely the presence, at its location and origin, of a group of embryonic cells identical to those of region ***f,*** cells which, in evolving, passed through the germination stage, visible in region ***n,*** then through that of region ***i,*** after having presented the appearance of region ***k,*** to end up in the state shown by the figure in ***a, b, b, b,*** a state which is not yet completely the caseous state, but which approaches it.

The reason for the formation of this caseous mass is not the necrosis of certain elements. The cause of the phenomenon which occurs here is totally different from that which causes, for example, the destruction of the inter-alveolar septum and epithelial cells.

Here, it is not a phenomenon of necrosis or involution that occurs, but on the contrary a phenomenon of evolution and multiplication. The evolution from the state of region ***f*** to the state of regions ***n,*** then ***k, i,*** and finally ***b,*** is a phenomenon of vegetation and the creation of new elements whose mass progressively increases.

The mass ***a, b, b, b***, is angular and not round, because the group of embryonic cells that constituted it already had this shape; but, through the development that will take place successively on all sides in the long run, the mass will necessarily round off and become a large spherical nodule.

Thus, Plate 1 has the advantage of showing us various very different aspects of the tuberculous infiltrating tissue in ***b, f, n, c, k,*** and ***i;*** it shows us that the two giant cells ***d, e,*** play no particular role in the development of the mass ***a, b, b, b,*** and that, evolving for their own account, like the other elements, they will come, like these, to be included in the nodular mass and to contribute to its formation.

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This shows us that, quite probably, the most frequent cause of the development of a nodule is the formation, at a specific point, of a mass of embryonic cells which, evolving fairly rapidly, becomes a nucleus around which new embryonic cells gradually form, develop, and evolve.

The mode of formation of the nodule of intra-alveolar origin is completely different, which will be discussed later.

Plate 30 shows us another aspect of tuberculous tissue; here, it is tissue gaining little by little by infiltration.

Pl. 30. *Section of tuberculous lung. Gross 335. Agr. 600.*

In this figure, we see a giant cell ***A,*** whose nuclei were roughly peripheral and in a crown. On the left and above, in ***B,*** we can see a zone of areolar tissue roughly similar to that of the giant cell; if region ***B*** did not take the form of a giant cell, it is because the embryonic cells were disseminated there, without order, and also less numerous than in ***A*** where their arrangement in a crown makes it appear as a giant cell.

Region ***C*** is very interesting because it also shows that tubercular tissue can take the form of reticulo-areolar tissue without taking the form of a giant cell. This tissue is formed, at ***a, b, c, d, e,*** by germinating embryonic cells, such as ***a, b, c, d, e, f, g, h,*** and also by the cells of row ***f,*** which has been detached from areolar tissue ***C*** by the section. At ***j,*** we see embryonic cells in the process of forming a small island of areolar tissue.

This clearly shows that the form of giant cells is governed solely by the arrangement and number of embryonic cells since, here, a simple line of confluent embryonic cells ***f, f,*** also gives rise to a characteristic mass of areolar tissue.

We will see shortly that these observations lead us to the explanation of the development of one of the forms of the reticulo-areolar tubercle.

Finally, **Plate 30** shows numerous embryonic cells in the process of germination. Two of them are seen in ***k*** and at ***l;*** in the latter, the granules give rise to filaments whose starting point is visible at ***m, m,*** for four of them; other groups of germinating cells are visible at ***n, o, p, q, r, s,*** and at other points.

I remind you that Plate 8 represents a field of the same preparation immediately adjacent to that of Plate 30 and that we see, at ***K,*** the germinative filaments of the embryonic cells ***L, M, N,*** and the beginning of the organization of these filaments into areolar tissue. I also point out that **plate 35**, which shows the formation of one of the types of intra-alveolar tubercle, represents both region ***H*** of plate 9 and region ***C*** of plate 30.

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The aspects presented by tuberculous tissue are so variable that a long series of forms could be given. Having already shown the main ones, I will limit myself to showing only two others in this description, because we will see even more new ones in the plates that follow.

Plate 31 represents a zone of infiltration tissue in which we still see a few young embryonic cells, but most of which are in the process of germination and giving rise to bundles of fibrils which, by anastomosing, come to form the most varied figures.

Chance is not the only factor involved in the formation of these figures.

Pl. 31. *Forms of tuberculous tissue. Gross 335. Agr. 600.*

The branches and fibrils that form these come from the germination of embryonic cells and, to be even more precise, from the large granules formed by them at the end of their development. We have seen that embryonic cells are an emanation of the supporting carcass or formative carcass of the inter-alveolar septum and are formed by its elements. Because of this origin, the embryonic cells and consequently the granules to which they give rise *have retained some of the properties possessed by the granules that formed the inter-alveolar septum.* The main property possessed by these was to organize themselves to form cellular elements, epithelial cells. We will see later that this is not a simple view of the mind but a fact t*hat finds its demonstration in the conservation of a part of these properties in these granules when, being separated from the organism, their multiplication is ensured on appropriate culture media.*

In **Plate 31**, the embryonic cells, through their granulations and the fibrils they emit, manage to form:

1. Irregularly shaped masses of tuberculous tissue, such as masses ***a, b, d,*** in which the constituent dumbbell-shaped elements can be distinguished, as in mass ***d,*** for example.

2. Curiously shaped figures, such as in ***c,*** for example, where the special arrangement of the dumbbell-shaped elements from the embryonic cells, represented by the small, dark black coherent masses, can be clearly seen.

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3. By giant cells such as ***f*** and ***e,*** the latter being small and not giant. They are both made up of very irregularly placed peripheral embryonic cells, which shows once again that the formation of the giant cell depends on the position occupied by the embryonic cells. The giant cell ***f*** here takes the form of an epithelial cell with its central nucleus at ***h;*** the dumbbell-like shape of the elements is very clearly distinguishable.

We will have the opportunity later to compare these formations of such diverse appearances with those observed in cultures of Koch's bacillus.

It now remains for us to examine what becomes of the elements of the tuberculous tissue, how they evolve to transform into caseous substance, and what form they possess once this transformation is complete.

We have already seen, by examining several figures, that the evolution towards the caseous state of the interior of the tubercle or of a region of infiltration tissue, results in the formation of a compact mass, in which one can less and less distinguish the shape of the elements which contributed to its formation.

We have seen in these figures, cited above, that the compact mass comes to be composed almost exclusively of dumbbell-shaped elements. The reason for this fact is that the embryonic cells, which are the almost sole formative element of the caseous substance

(with debris of degenerated epithelial cells), evolve to form large granules **(1)**; when the cell has completed this evolution, these are set free by the simple fact that the cell itself no longer has an envelope and no longer exists; then they germinate to give rise to new dumbbell-shaped elements or to filaments constituted by series of rods and dumbbells.

However, when the contents of a cavern, large or small, are removed, a large quantity of embryonic cells and epithelioid cells are found in the caseous matter. This is because, when a cavity is formed by the elimination of caseous matter from the outside, this elimination does not only affect the latter; it affects elements whose development is not complete, and especially among these, embryonic cells.

The caseous transformation of tuberculous tissue does not always completely eliminate its areolar form; areolar voids may persist, formed by large anastomosing trabeculae; plate 32 gives us an example.

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This is a case of tuberculosis in a miner, Michel, whose study elements, fully prepared, I owe to the kindness of **Dr. Ameuille,** whom I thank for the very interesting materials he was kind enough to make available to me.

The figure represents a region bordering a small cavern ***a;*** we see, in ***f,*** the areolar voids, in ***b, g,*** the trabeculae which delimit them, and in ***c, d, g, h,*** points where the trabeculae, having reached the end of their evolution, dissociate, to allow the granulations and fibrils which they contain to escape into the areolar cavities. In the entire region between points ***A*** and ***B,*** we see only rare embryonic cells ***q, r, s, t,*** for example, and a few elements such as ***k, l, m, p,*** which seem to me to be able to be only epithelioid cells. In fact, what we see here is a tuberculous tissue whose trabeculae, having reached the caseous state, contain almost exclusively dumbbell-shaped elements and granulations.

I will conclude this presentation by showing, through **plates 33 and 33a**, how the germination of embryonic cells comes to form the large trabeculae of tuberculous tissue, such as those shown in **plate 32.**

These two figures represent photographs of two fields immediately adjacent to the one shown in figure 1 of plate 3 and which shows, in the same subject and in the same lung section, the origin of embryonic cells.

In **plate 33,** which is a very thin section and very favorable to observation, we notice large trabeculae such as ***a, c, d, e,*** of very clearly fibrillar constitution; we see, in their interior, barely colored granules; in some points we see a filament starting from a granulation, in others, very clear dumbbell-shaped elements.

(1) *It will be demonstrated later that these themselves belong to dumbbell-shaped elements.*
Pl. 32. *Forms of tuberculous tissue. Gross 335. Agr. 600.*

In ***b,*** we see a group of well-colored granules, which emit filaments that engage in trabeculae ***a*** and which are dumbbell-shaped elements, which is visible for three of them. This group of granules is a mature embryonic cell, which germinates to constitute, at least in good part, trabeculae ***a.*** In ***c,*** the granules of another embryonic cell also emit dumbbell-shaped elements that engage in the trabeculae.

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Cell f is formed of large granules which emit in ***j, l, s, t,*** dumbbell-shaped germinative filaments; cell ***h*** also emits some, three of which are visible at ***v, u.***

The trabeculae ***G*** and ***F*** are both made up of parallel dumbbell-shaped filaments that emanate very visibly from the granulations of two embryonic cells located below the letters ***G*** and ***F.***

Note that the entire part of **plate 33** located above the letter ***B*** is reproduced in **plate 33bis** with a different focus, from the lower part of the latter to the letter ***P.*** This different focus makes it possible to see very precisely in the region ***R, T, A, B, C, D, E,*** the constitution of the trabeculae by dumbbell elements.

All the filaments visible in the figure are formed by dumbbell elements. A good number are designated by a cross.

Cell ***x*** emits a dumbbell-shaped filament ***A*** which continues with two others ***A′, A″.***

Large trabeculae branch off from smaller, highly branched trabeculae, such as ***o, p, q, r,*** which constitute the areolar tissue in formation.

Plate 33bis also shows a group of several sinuous, branched, anastomosing trabeculae ***a, b, c, h, g, k,*** consisting of fibrils and granulations. By looking with a magnifying glass, and even with the naked eye, it will be clearly seen, at several points, that the fibrils are made up of dumbbell-shaped elements placed end to end, for example at ***p, q, t, p.*** At ***q,*** a granulation will be seen emitting a long fibril, made up of 3 or 4 dumbbells.

We can also see, in the figure, embryonic cells at all stages of development, for example a young one at ***d*** with its pedicle and several others at ***1, 2, 3,*** one at ***r,*** with its formative dumbbell ***rr″*** at ***s.*** A large cell with a short pedicle which is a dumbbell element, another at ***e*** formed by the dumbbell ***f;*** at ***x,*** we see two large cells at a more advanced stage and more voluminous, then at ***v,*** two cells which have reached the end of their development and whose very numerous granulations are almost free; some of them are beginning to emit a germinative filament.

The most remarkable part of this figure exists at its lower part and mainly at the level of the areoles ***A, B, C, D, E, F,*** formed by the very developed trabeculae which already represented in its central part **plate 33.**

In this region, we can clearly see the shape of numerous dumbbell-shaped rods, as well as the arrangement of their assembly to form the trabeculae that delimit the areolar voids.

We will see that the dumbbell rods are arranged in parallel in groups of 2, 3, 4, 5, 6, etc., by placing their granulations against each other, since they are arranged end to end with others by also placing their granulations against each other in such a way that at the junction point of two dumbbells, there are two granulations placed side by side.

We will be able to see in ***H I*** and especially in ***J K L,*** then in ***M N***, young trabeculae in the process of formation grouping only two or three dumbbell rods or filaments. The shapes of the dumbbell rods, the arrangement of their assembly to form the trabeculae of the tuberculous tissue, that is to say this tissue itself, are, here, so clear that I consider these points as definitively established.

This demonstration of the constitution of the trabeculae and the arrangement of the elements or fibrils in a dumbbell shape has already been given elsewhere in connection with the explanation of **Plate 29.**

These **Plates 33 and 33bis** therefore provide new demonstrations which confirm:

1. The origin and nature of embryonic cells,

2. That all the phases of their evolution take place at the very point where they are born.

3. That at the end of their evolution, they become a mass, a veritable bag of granulations.

4. That at the end of this evolution, these granulations germinate and give rise to dumbbell-shaped rods.

5. That the trabeculae which constitute the framework (or intermediate matrix) of the tuberculous tissue are the result of this germination and that they are formed exclusively by its products, that is to say, by dumbbell-shaped rods and filaments composed of these rods.

* * *

We saw in **Plate 32** that the tuberculous tissue resolves, at the end of its development, into a mass of granulations and filaments or dumbbell-shaped rods which fall into the areolae of the tissue, thus constituting cavities which, when combined, will constitute a cavern. The study of Figure 1, **Plate 34** will confirm the previous observations on the constitution of the caseous matter of tuberculous lesions of the lung.

Pl. 33. *Formation and constitution of the intermediate frame. Gross 550. Agr. 1,000.*
Pl. 33 bis. *Formation and constitution of the intermediate framework. Gross 550. Agr. 1,000.*

This figure shows what is seen in old caseous matter whose development is complete, taken from a cavern, spread on a slide and stained with Ziehl's fuchsin.

We realize, first of all, that it is mainly made up of granules of various sizes and dumb-bell-shaped rods; we see, in certain places marked by a cross, rare embryonic cells, with very apparent contours, not completely developed; in other places, we can no longer distinguish anything but the outline of these cells, as in m, for example; but, on the other hand, we can clearly distinguish the granules that they have formed.

Throughout the figure, we can see dumbbell-shaped rods formed by granulations of various sizes; one of them ***b*** emits a granular dumbbell-shaped rod ***c,*** resembling a Koch bacillus; we will have the explanation of this fact later.

Attention being drawn to the fact that all these filaments are, in reality, dumbbell shaped elements, we can quickly see that the entire caseous matter appears to be made up of these dumbbell-shaped elements.

By looking in the figure, we find quite long, divaricated and branched fibrils, bearing young, short-pedunculated granulations such as fibril ***f;*** but it appears that these small granulations themselves belong to small, developing dumbbell-shaped elements.

Certain dumbbell-shaped elements, such as ***k, l, d, e,*** are formed by two granulations of unequal size, one highly colored, rich in chromatin, the other barely colored and very refractile.

These various observations can also be made in the figures in **plates 47 and 48**.

Let us recapitulate the facts acquired by the successive study of the embryonic and giant cells of the framework of the tuberculous tissue, its various vegetative forms, and its evolution.

1. The giant cells emit expansions whose ramifications penetrate between the cellular elements. These expansions are themselves made up of elements resulting from the germination of the peripheral nuclei of giant cells, nuclei which, in reality, do not differ in any way from embryonic cells.

2. Embryonic cells germinate and form fibrils and dumbbell-shaped elements grouped into bundles that anastomose with each other and form an intermediate framework or matrix that encompasses all the cellular elements and, with them, constitutes the tuberculous tissue.

Pl. 34. *Fig. 1. Constitution of caseous matter. Gross 1,100. Agr. 2,000. Fig. 2. Evolution of embryonic cells. Gross 550. Agr. 1,000.*

3. Tuberculous tissue occurs in three main forms.

A) The form that corresponds to the birth and development of embryonic cells and is, most often, that of vast areas where cells emerging from fine filaments or branched bundles abound.

B) The form that corresponds to the germination of embryonic cells and which, in this case, occurs in the most diverse aspects, but which always amounts to a tissue formed from anastomosing bundles.

C) The terminal form, which evolves towards caseification and in which the tuberculous tissue no longer presents anything but rare and large lacunar spaces, the tissue tending to become compact and comprising almost nothing more than unorganized granulations and fibrils as elements.

4. The giant cell is a localized point in the vegetation of the tuberculous tissue, whose development and evolution do not differ in any way from the tissue of neighboring regions; its form is governed only by the original situation of the embryonic cells that constitute it.

5. The caseous matter is constituted exclusively, at the ultimate end of its evolution, by dumbbell-shaped elements; when it still contains embryonic and epithelioid cells, it is because the evolution of these elements is not complete.

6. The caseous matter, the terminal result of the evolution of the tuberculous tissue, does not result from a process of necrosis, but from a phenomenon of vegetation and multiplication of the constituent elements of the embryonic cells, the chromatic or nuclear granules and their dumbbell-shaped rods.

7. It has been shown, with the study of the constitution of the epithelial cells and the supporting framework of the inter-alveolar septum, that the fundamental and unique element which serves to build the framework and the entire organization of the cell, the organs and the totality of the organized being, is the dumbbell-shaped rod; it is this rod, originating from the epithelial cells and other elements of the pulmonary tissue, which generates the embryonic cells. These, instead of fulfilling their role of repairing the epithelium, become the agents of the multiplication of the dumbbell filaments; these, in turn, become the agents of tuberculous infiltration and the destruction of the tissues. The final state of this destruction is the caseous matter and the dumbbell-shaped rod which constitutes it.

Thus, when the organization of a tissue and its anatomical elements is destroyed, the fundamental elements that constitute them remain alive and multiply, but they no lon-

ger obey the laws of organization. Just as with tissue, when the entire organism dies, the fundamental elements that constitute it remain alive and continue to multiply.

This evolution is the fate of all living organisms affected by organic death; it is that of the body of all animals, and the waxy appearance that their tissues eventually take on, the cadaveric fat, results from a transformation identical to the caseation of tubercular tissue. The organization dies, but the fundamental elements that constitute it do not die and multiply or renew themselves as long as the environmental conditions are favorable.

CAUSES OF THE NODULAR SHAPE OF TUBERCULAR TISSUE

The development of tuberculous tissue around one or more neighboring giant cells, or the fact that the nodule includes a group of pulmonary alveoli, the cavities of which are filled with desquamated epithelial cells, have been accepted as the principal causes of the rounded shape of the tuberculous nodule and as the cause of its formation.

In certain rather rare cases, the center of the nodule is occupied by one or even two or three giant cells not far from each other; this does not mean that it was the giant cell that was the starting point for the formation of the nodule; this starting point is the entire mass of embryonic cells in which the central giant cell was formed; it is the germination of this mass of embryonic cells that forms the tubercle.

It has been shown above that, in reality, it is a region of the infiltrating tissue, a small zone of embryonic cells whose development is more advanced than the neighboring ones, which begins to germinate and constitutes the center of the nodular mass or even its oldest part.

This formation is due to a cause analogous to that which determines the formation of a giant cell, the particular arrangement of a group of embryonic cells in germination.

To understand these facts, it is necessary, of course, to examine young tubercles at the beginning of their formation.

There are other cases where the nodular form of tuberculous tissue is caused by the formation, in an alveolar cavity, of tuberculous tissue which fills it, by vegetation of the desquamated cells and by the germination of embryonic cells born in great numbers on the alveolar walls.

Plate 35 shows an example of this development; the alveolar cavity, a, contains the debris of desquamated epithelial cells which vegetate and form young embryonic cells ***b, c, d,*** for example or older, ***q, s, t, u, v, x, y, z,*** whose vegetation will fill the alveolar cavity with tuberculous tissue.

The embryonic cells of the intra-alveolar wall send germinal bundles into the alveolar cavity, which branch out there, forming areolar tissue; parts ***e, f, g*** have this origin, and come from the alveolar walls ***h*** and ***i*** from which they were detached by the preparation maneuvers.

In addition, certain branches emanating from the wall, such as ***m, n***, at the bottom and left of the alveolar cavity, ***p,*** at the top of this cavity, contribute by their vegetation to the formation of tuberculous tissue in the alveolar cavity.

A second example of the formation of an intra-alveolar nodule is provided by **Plate 36**. In ***a, b, c, u***, we see what remains of the inter-alveolar partitions, rich in embryonic cells, a certain number of which appear with their formative pedicles. The intra-alveolar mass, already consisting of bundles intertwined in all directions, contains debris from a certain number of epithelial cells ***m, n, o, p, q, r,*** for example, debris which are vegetating; proof of this is provided by the degenerating epithelial cell ***r,*** which emits two bundles branching into set ***t.***

Part of the nodular mass is formed by vegetation coming from a group of epithelial cells from regions ***d, e, f, g, h***, mainly; the tissue from regions ***h, l,*** comes from the germination of the embryonic cells of the wall.

Numerous embryonic cells are formed or are in the process of forming throughout the intra-alveolar mass; they are marked with a cross at the end of the indicator line or simply with a cross.

Finally, I will give, with **plate 37**, a last example of an intra-alveolar formation which shows, moreover, one of the typical aspects of the vegetation of tuberculous tissue and, moreover, the part that the inter-alveolar septum can play in this vegetation.

In ***A*** we see an infiltrated inter-alveolar septum rich in embryonic cells in very clear germination; in ***E,*** a crossroads from which a septum ***B*** started on the right and another ***C*** on the left, the alveolar cavity being completed by septum ***D.***

From partition ***C,*** the bundles ***b, c, d*** branch off, which will branch out to the center of the intra-alveolar mass; from partition ***B,*** the bundles ***e, f, g*** branch off; from partition ***D,*** several others branch off; one is designated ***h, g.***

Pl. 35. *Formation of the tubercle of intra-alveolar origin. Gross 335. Agr. 600.*
Pl. 36. *Formation of the tubercle of intra-alveolar origin, Gross 335. Agr. 600.*
Pl. 37*, Formation of the tubercle of intra-alveolar origin. Gross 335. Agr. 600.*

By examining the figure, one can easily see the great importance of these bundles in the formation of the intra-alveolar mass.

Numerous embryonic cells are born on the branches of these bundles, whose germination will eventually fill the empty spaces.

In addition, the germination of embryonic cells of the inter-alveolar septa will complete the formation of tubercular tissue at the periphery, as, for example, in region ***a;*** germinating embryonic cells can be seen almost everywhere, but especially in regions ***A, E, j, k, l.*** The intra-alveolar mass also contains debris of epithelial cells, which must also have contributed significantly to the formation of the intra-alveolar mass. These are seen in ***n, o, p, q, r, s.***

Note, in this figure, the very special form that the vegetation takes in regions ***t, u, v,*** and which has already been observed in other figures, for example, in **plate 31**; it will be seen, by examining this region ***t*** and its periphery, that its particular appearance is due to a special arrangement of the elements in a dumbbell.

This plate also provides a very clear demonstration of the origin of embryonic cells; by examining them with a magnifying glass, the reader will see them of all sizes and in all stages of development, clearly emerging on their formative branch; some are equipped with the dumbbell which constitutes their formative pedicle.

* * *

In summary, these nodular formations result from:

1. Vegetation, in the alveolar cavity, of the debris of degenerated epithelial cells.

2. The germination of embryonic cells born on this debris.

3. The vegetation of the branches emitted by the inter-alveolar septa and the embryonic cells they form.

4. The germination of embryonic cells born on the walls of the inter-alveolar septa.

At this point, we have already successively investigated:

1. The origin and nature of embryonic cells.
2. The origin and nature of desquamated cells in the pulmonary alveoli.
3. The constitution of epithelial cells.
4. The constitution of the framework or carcass of the inter-alveolar septa.

5. The destiny or evolution of desquamated epithelial cells.
6. The evolution of the framework of the inter-alveolar septa.

7. The constitution, nature, and origin of giant cells.
8. The existence and constitution of the intermediate framework of tuberculous tissue.
9. The various forms of tuberculous tissue.
10. The determining causes of the nodular form.
11. The evolution of tuberculous tissue from the birth of the cells embryonic to the formation of caseous matter, and we have determined its constitution.

Now, throughout this study, we have witnessed the destruction of lung tissue and we have not yet been led to deal with Koch's bacillus or to perceive its role.

Before studying this role, there remains one last question to study: it is the constitution of embryonic cells; we have already, without specifically researching them, acquired notions of capital importance on the evolution of these cells; we will seek, at the same time, to complete these notions, then we will study the constitution, the nature and the role of an element of which we have not yet spoken: *the epithelioid cell.*

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CONSTITUTION AND EVOLUTION OF EMBRYONIC CELLS

I will briefly recall here all the new knowledge we have already acquired about embryonic cells.

1. They are born:

A) On the carcass of the inter-alveolar septum in the process of degeneration and on the epithelial cells themselves, still in place.

B) On the debris of desquamated epithelial cells that have fallen into the alveolar cavities, particularly on their frames. We have seen that this debris remains alive and that their constituent elements remain capable of reproduction and the formation of embryonic cells.

C) On the branches emanating from the inter-alveolar septum.

D) On the filaments originating from germinating embryonic cells and on the framework formed by these filaments grouped into bundles and which constitutes the tuberculous tissue.

2. At their birth, they are tiny, only the size of a granulation, then grow until the end of their development.

3. They are sometimes sessile, but generally formed at the end of a pedicle, often club-shaped, consisting of one or two dumbbell-shaped elements.

At the end of their development, the granulations they have formed germinate, emitting filaments which, by joining with others, constitute the bundles and areolar framework of the tuberculous tissue.

We have already presented numerous facts relating to the development of embryonic cells. We will supplement them with new observations.

Figure 2, **plate 34** shows in ***e, f,*** two medium-sized embryonic cells and elsewhere, in ***a, b***, then especially in ***c, d, h,*** cells with completed development, full of very large granulations, of which one ***d,*** has the appearance of a raspberry; its pedicle ***g*** arises on the disintegrated alveolar wall ***i.***

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Figure 1, **Plate 38** shows embryonic cells in the process of development, some of which are undergoing a development that has not been discussed so far. The peripheral zone of young cells, which has been described as the peripheral protoplasmic zone, increases considerably in thickness and thus forms the cell described under the name of plasma cell; the figure shows, at ***l,*** a normal embryonic cell with its pedicle ***m,*** then, at ***j,*** three other normal cells, consisting almost exclusively of a nucleus without an appreciable external zone or envelope; at ***a,*** we see a cell in which this external zone has already become very clear, then another at ***b,*** whose external protoplasmic zone ***q*** is quite notably developed; the same is true for cells ***e, f.*** In cells ***c, d,*** the external zone has taken on a large extension; finally, we see at ***g, h, i,*** a group of several cells in the process of merging to form an epithelioid cell which will perhaps even enclose cell ***q.***

Let us point out the germination of the embryonic cell ***r,*** whose granules form the filaments that constitute the areolar tissue of the trabeculae ***s, t,*** connected to the trabeculae ***u*** and surrounding the cavity ***v.***

Let us also note that, in the cell ***q,*** we see a granular filament ***o*** resembling a Koch bacillus and which bears, at its two ends, a refractile granule ***b;*** it is therefore a dumbbell-shaped element.

Finally, the embryonic cell ***A*** releases its granules on one side, some joined together in a dumbbell by a rod such as ***z*** and ***y s.***

Thus, Figure 1, **Plate 38** shows us the evolution of the embryonic cell into an epithelioid cell, by the formation, around it, of a zone of elements less rich in chromatin.

We will see later that embryonic cells contain both chromatic granules and non-chromatic granules, that is, granules that stain only very weakly and are very refractile.

Pl. 38. *Evolution of embryonic cells. Fig. 1: Grosst 700. Agr. 1,250. Fig. 2, 3, 4, 5. Grosst 1,100. Agr. 2,400.*
Pl. 39. *Evolution of embryonic cells. Gross[1]. 700.*

Only a small number of embryonic cells undergo the transformation into epithelioid cells. The latter are not formed exclusively by this process; they are also formed by the fusion of several embryonic cells; Figure 1, **Plate 38**, which we have just explained, shows an example of this fusion in ***h, i, y.***

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Plate 39 gives new and multiple examples of this formation of epithelioid cells by fusion. In all the figures in this plate, we will see embryonic cells that group together in threes or fours to fuse and form globular, round epithelioid cells, such as those in Figure 8; more rarely, embryonic cells fuse in a line, as in Figures 3, 5, and 7, which show examples, to form very elongated, straight or curved epithelioid cells, like those in Figure 9. On the far right of Figure 7, we see an arcuate epithelioid cell, in formation, in which the formative embryonic cells can still be seen.

It has been previously demonstrated that the usual destiny of embryonic cells is the germination of their granulations and the formation of trabeculae of tuberculous tissue, then the transformation into caseous matter. But, if we remember that embryonic cells are born by budding on the inter-alveolar septum, and that we have considered this budding as constituting, evidently, the beginning of the process of repair of the epithelium, we should no longer be surprised that some of these embryonic cells are transformed into cells so resembling epithelial cells that they have been named epithelioid cells.

The latter, "having exclusively their origin in embryonic cells," as **Hollande (1)** saw, are not lymphoid productions, as he also affirmed, since embryonic cells are formed on the spot by various elements of the inter-alveolar septum and, consequently, have nothing in common with lymphoid elements.

* * *

Plate 40 provides us with new information on the phenomena which lead, within the embryonic cell, to the formation of granules.

By examining the embryonic cell in **plate 40,** we see it constituted by a coiled, very sinuous chromatic filament, 1, 2, 3, 4, 5; at first glance, this filament appears to correspond to the spire stage of a cell in the process of cytokinesis; it appears segmented in places, at the level cell in the process of cytokinesis; it appears segmented in places, at the level of the refractile granules, so as to form arcuate or ***U-***shaped dumbbells, consisting of a chromatic rod bearing, at each end, a colored or colorless and refractile granulation, that is, chromatic or deprived of chromatin; the dumbbell 2 of cell ***a,*** arched in a ***U,***

(1) *A. Ch. Hollande and Mrs. G. Hollande,<Histogenesis and Cytology of the Tuberculous Follicle>, Arch. d'anat. micr., 1931, p. 1.603.*

Pl. 40. *Constitution and evolution of embryonic cells. Gross 1,100. Agr. 2,000.*

has, for example, a colored ball at one end, an uncolored one at the other. The proof of the formation of rods into a dumbbell is clearly given by cell ***d,*** **plate 40** and by cell ***a*** of **plate 41**; this last cell shows four parallel, strongly colored chromatic rods, bearing at each end the small ball which gives them the dumbbell shape.

I do not wish to dwell further on this phenomenon of multiplication of granules in embryonic cells, because if the examination of cells ***a, b, c, d, e, p,*** **plate 40** and ***a, b, c, d, f, g, h,*** **plate 41** allows us to affirm that they are in a state of chromatic division and multiplication of their granules, none allows us to specify very clearly the details of this multiplication.

If we recall that it has been shown, in numerous previous plates, that embryonic cells are tiny at the beginning and begin their evolution in the state of a simple granule, we are led to conclude that it is this continuous multiplication of the primitive granule that results in the formation of the adult embryonic cell constituted exclusively by a group of granules.

Examination of many embryonic cells, in almost all the preceding plates, shows that the fully developed cell contains both highly colored chromatic granules and non-chromatic, highly refractile, colorless granules, because they have no affinity for Ziehl's fuchsin.

Figures 2, 3, 4, 5, of **Plate 38,** show, at a very high magnification, the final state of mature embryonic cells in the caseous substance of a cavern.

Large chromatic granules, black in the figures, and refractile, colorless granules can be seen. These two types of granules are very apparent in embryonic cells ***a, e, k,*** Figure 2, ***a, b,*** Figure 3. In Figure 5, we will see a mature embryonic cell having the appearance of a raspberry and, in the upper left part of the cell, a short dumbbell-shaped rod whose granulation on the right is barely visible, that on the left even less so.

In Figure 4 of the same plate, we see an epithelioid cell in the process of formation by radiating dumbbells starting from the central nucleus which was a simple embryonic cell.

Plate 41 represents a region quite rich in embryonic cells. Among these, a certain number are already germinating to form a very large-mesh areolar tissue that can be seen at all points in the figure. This areolar tissue will provide us with the subject of some observations that complement those we have already made previously on the same subject.

In this **plate 41,** in the inner quarter and towards the middle part, we see, at ***r, a*** thin filament which starts from a span and is made up of two others emitted by two barely visible refracting granulations, ***s, t,*** which form, at ***u, j,*** a group of eight or nine small granulations, some colored, ***u, j,*** others ***m,*** uncolored, very refracting. From these granulations, filaments start; for example, granulation ***j*** emits a filament which ends in granulation ***k.*** The large refractive granulation itself emits a filament carrying a barely visible uncolored ball which in turn emits filament ***n,*** which ends in the *slightly colored* granulation ***o;*** in turn this emits a new, very barely visible filament ***p,*** which appears to end in the ball ***q.***

We will also see, in a small span of **plate 36,** on the right and below, the same filament ***z*** joining granulations ***z'*** and ***z".***

By examining the small spans, in the various regions of the figure, we will see them all constituted exclusively by dumbbell-shaped filaments of varying length.

We have already seen in **Plates 24 and 33** that four or five successive dumbbell rods can be assembled end to end to form fibers which, joined in parallel by 2, 3, 4, or more, constitute the trabeculae of the tubercular tissue. I believe that these dumbbell fibers can, in reality, be much longer.

It appears that certain fibers are not always made up of dumbbell rods placed end to end, but that they have, at least in places, the appearance of knotted rope filaments, that is to say, at the point where the rods join there is sometimes only one ball instead of two.

Thus and by this process, the tissue is formed which forms the trabeculae of the areolar tubercular tissue; it is enough to examine this tissue, in ***A, M, N, I,*** **Plate 41** and everywhere, in all the plates and with a magnifying glass, to be convinced that it indeed has this constitution.

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This process of formation, by the dumbbell-shaped elements of the trabeculae of the tuberculous tissue and also of the areolar tissue of the giant cells, is also the one that constitutes the normal anatomical elements of organisms.

This process of formation, by the dumbbell elements of the trabeculae of the tuberculous tissue and also of the areolar tissue of the giant cells, is also that which constitutes the normal anatomical elements of animal and plant organisms.

Proof of this is provided by the examination of plates 14, 15, 16, 17, 18, 19, showing the constitution of the epithelial cells of the interalveolar partitions of the lung and of the

Pl. 41. *Constitution and evolution of embryonic cells. Gross 1,100. Agr. 2,000.*

carcass of these. This is the fundamental process of construction of all tissue and of all organized beings. I wanted to convince myself of this by extending my observation to the most weakly organized living matter, a filament of mold, of mucor for example.

Plate 57 shows the constitution of such a fiber and the fundamental element of its organization, the rod or dumbbell filament; this question is treated on page 137.

It is shown here that molds are still organized beings, however weakly; living matter reduced to its simplest expression and totally devoid of organization is the bacterial culture of granular form, that of a micrococcus, for example. The expression tuberculous tissue, which I have used until now, is therefore quite accurate; it is indeed a tissue which replaces the destroyed normal pulmonary tissue, but it is an abnormal tissue, whose formation no longer obeys the rules of the organism and whose final state is the caseous state.

It remains for me to show some other important facts contained in plates 40 and 41:

1. The vegetation of embryonic cells to form the tuberculous tissue of the trabeculae.

The formation of the trabeculae will be observed by the germination of the cells ***e, v', x, y, z,*** **plate 40**, and ***B, C, D, E, F, G, H, J***, **plate 41.** As the filaments and trabeculae develop, the embryonic cells become less and less visible; only their outline can be distinguished, as in ***E, F, H, G,*** **plate 41**; then they finally disappear completely; at the same time, the trabeculae continue to widen, until they completely fill the areolar voids and form compact masses of caseous matter.

The germination of embryonic cell ***C***, **plate 41**, is particularly remarkable; the large trabecula ***C',*** which it forms, is visible only at its origin; the radiating dumbbell filaments that the cell ***C*** emits at its periphery are very apparent; the same is true for embryonic cell ***v',*** **plate 40,** which forms trabecula ***v.***

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2. Many embryonic cells are clearly equipped with their formative pedicle, for example, cells ***f f', hh', K K', L L', d, a, y, y'***, **plate 41**, etc.

3. We note in **plate 35** the presence of four embryonic cells, of a special form ***r, s, t, u;*** they are elongated and we do not see a well-defined nucleus as in epithelioid cells; these four cells are each equipped with their very apparent formative pedicle, ***k, t', s', u'***; I think they are epithelioid cells. They are formed by dumbbell-shaped elements quite clearly visible. **Ach.** and **G. Hollande** describe in the evolutionary cycle of the tubercle bacillus, four different forms of which the first is a very fine basophilic granulation, the size of which would be at the limit of microscopic observation; the second would be a dumbbell shape, the third a non-acid-fast bacillus and the fourth, the normal acid-fast bacillus.

Regarding the second stage of this evolution, they say, "*We have exceptionally seen two of these granules (those of the first stage) located side by side, reminiscent of a fine diplococcus; we thought that this was the subdivision, into two elements, of a primitive granule. We base this opinion on the fact that it was possible for us to observe, on several occasions , and with all desirable clarity, in the protoplasm of some epithelioid cells, the presence of a very tenuous filament, connecting two similar granules, that is to say, basophilic*."

Further on, they add: "*the second stage of the evolutionary cycle of the tuberculosis virus is thus constituted by an imperceptible filament presenting a tiny basophilic granule at each end*."

These details clearly show that the dumbbell shape seen by **A.-Ch**. and **G. Hollande** is an exceptional form and not the characteristic normal dumbbell element which constitutes the entire epithelioid or giant cell, and whose end granules have a diameter of 0.4 to 0.7 or 0.8 m on average and always 2 to 3 times larger than the width of the rod which connects them. I have never seen almost imperceptible dumbbells; there are thin ones and larger ones, but they are all clearly visible. This dumbbell element has already been shown so many times in the preceding plates that it is unnecessary to dwell on its dimensions here.

According to **A.-Ch.** and **G. Hollande**, the presence of an imperceptible dumbbell shape in epithelioid cells would be an inclusion of a special stage of the tuberculosis virus, which would be carried out to destroy it.

We will see later that this hypothesis is not correct, because the dumbbell-shaped rod, which is the fundamental constituent element of epithelioid cells and giant cells, is at the same time Koch's bacillus itself; it is because this is the normal constituent element of these cells that it is found in their interior, and not by virtue of the phenomenon of phagocytosis, which has been invoked to explain this fact. Thus, the main explanation given to prove the existence of the phenomenon of phagocytosis is demonstrated to be incorrect; this phenomenon does not exist.

Following the many observations we have made, either in this chapter or in the previous ones, we can establish the origin and development of embryonic cells as follows.

The origin of an embryonic cell is one of the balls of a dumbbell-shaped rod; it is because of this origin that the embryonic cell is so small at first. This ball of the dumb- bell-shaped rod multiplies, forming new dumbbells, which constitute embryonic cells such as ***a, b, c, d,*** **Plate 40;** ***a, c,*** **Plate 41.**

The pedicle of these cells appears to be club-shaped because it is formed by a dumbbell of which only the rod and the external ball are visible, the second ball being included in the cell, at the very point of insertion of the rod.

The appearance of certain embryonic cells such as ***a, d,*** **Plate 40,** and ***c,*** **Plate 41,** appears to show that they are formed by a dumbbell filament arising from the original ball and winding into a spiral to form the external part of the cell. The final appearance of evolved cells, as presented in Figures 2 to 5, **Plate 38,** for example, shows that the balls of the dumbbells grow considerably larger or fuse together to form larger ones. The germination of the ball of a dumbbell to form an embryonic cell, then the germination of the dumbbells or granulations of the dumbbells of the latter, when it has evolved, to form new dumbbells which themselves constitute a tissue or an anatomical element, is the general phenomenon which regulates the formation of the latter; it is obviously this phenomenon which, at the very origin of the development of the embryo, presides over the first cellular multiplications. I conclude from this that it is very probable that, in all the phenomena of cytokinesis, the chromosomes are dumbbell-shaped elements; It also seems probable that the multiplying cells must form in the embryo by the process just indicated: the germination of one of the balls of a dumbbell-shaped element.

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NATURE AND ORIGIN OF KOCH'S BACILUS

Research into the origin of Koch's bacillus in tuberculous tissues.

To understand the origin and role of Koch's bacillus, it is necessary to study the relationships it presents either with the normal elements of lung tissue or with the elements of tuberculous tissue, as we have described them so far.

I had great difficulty in carrying out this study because, to determine the presence of the tuberculous bacillus in the sections, it is necessary to decolorize them, by immersion in a quarter-strength sulfuric acid solution, for example, and thus the elements other than the bacillus become almost invisible; to be able to appreciate the relationships between them, it is necessary to recolor the section.

The process which proved most favorable and which allowed me to obtain the photographs of **plates 42, 43 and 44**, consists, instead of recoloring the section, in not decolorizing it completely; To do this, it is gradually decolorized, in several stages, with a quarter of sulfuric acid, after having first stained it strongly with Ziehl's fuchsin. The section is examined under a microscope after each operation, and the decolorization is stopped when, the tissues being still sufficiently stained to allow observation and photography, the Koch bacilli remain brightly colored and stand out against the background.

This study was made easier for me by **Dr. Ameuille**, a hospital doctor, whom I thank here, and thanks to whom I was able to study, in particular, a case of tuberculosis in a miner (Michel) which lent itself particularly well to the research undertaken. The three **plates 42, 43 and 44** relate to this case.

Here are the facts that could be observed with the help of these materials: These facts are of several kinds and relate to:

1. The shape of the tubercle bacilli.

2. To their coloring, or rather to their acid resistance and the cause of it.

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1. Shape of Tuberculosis Bacilli in Sections.

Let us examine in **Plate 42** the bacilli marked with a cross, particularly bacilli ***a, b, c, d, e, f,*** and in Figure 1 of **Plate 43**, the bacilli marked ***a, b, c, d, e, f, g, h, i, j, k, l, m, n, o.***

This examination teaches us:

A) That the intensity or degree of coloration of the bacilli is variable.

B) That some have a uniform coloration and do not have a granular appearance, while others, such as ***f,*** **Plate 42**, are distinctly granular.

C) That all the bacilli consist of a more or less long rod, straight or arched, bearing a ball, generally clear and very refractile at each end; they all have the shape of a dumb-bell.

D) By searching the plate, we note the presence of numerous small, barely colored rods, also bearing a refractile ball at each end; they are identical to the colored bacilli and, like them, have the shape of a dumbbell; they differ from them, strictly speaking, only by their lack of coloring, that is to say, acid resistance.

In Figure 1 of **Plate 43** and in Figures 1, 2, and 3 of **Plate 44**, we can make the same observations. The bacilli all have the shape of a dumbbell, and their degree of coloring is variable. In Figure 2, **Plate 44**, we see in ***a*** a typical Koch bacillus, granular, in the shape of a dumbbell, with its two granulations, in b and c.

Figure 4, **Plate 44** shows a group of embryonic cells in another case of tuberculosis; all the cells on the left side of the figure are formed by dumbbell-shaped rods, such as ***h, o, q, r, s, t, u,*** and identical to the isolated dumbbell ***y.***

Whether strongly stained with acid fuchsin or unstained, the bacilli are identical, and staining is not a characteristic that can serve to differentiate them; their different degree of staining is due, not to their being of a different species or origin, but to the fact

that a slow modification is taking place in them, corresponding to a progressive diminution of the substance in them which has the affinity for acid fuchsin, which is chromatin.

This explanation is justified:

1. By the fact that all degrees of coloration of the bacilli are found, from the most intense to almost total absence, the bacilli being almost no longer visible at this time except thanks to their refraction.

2. By the fact that the chromatin does indeed disappear from the bacilli as they age; they are first a full and continuous rod of chromatin; then it gradually leaves the bacillus, which corresponds first to the formation of clear transverse striations; later, as these increase in width, the chromatin is reduced to three or four granules, which are Much's granules.

This evolution of the chromatin can be easily observed by anyone examining the bacillary forms, from the large rod, Koch's bacillus full of chromatin, to the clearly granular bacillus on the way to becoming uncolorable; this observation can be made in **Plate 46.**

Figures 2 and 3 of **Plate 43** show us a new fact of capital importance. These two figures represent the same region photographed with two different microscope focuses. In Figure 2, we first note, as before, the dumbbell shape of the bacilli ***a, b, c, d, e, f, g, h, i, j, k, l, m, n, o, p***, straight or arched and more or less strongly colored; but in addition, we notice a group of bacilli ***e, f, g, h, i, j, r,*** some of which are arched and we see, moreover, that these bacilli or acid-fast rods are grouped in clusters because they are contained in an embryonic cell ***A***, the outline of which is marked by a dotted line in Figure 3. In this last figure, certain bacilli of the group, not visible in Figure 2, have been brought into focus.

In Figure 4, **Plate 42,** we see an embryonic ***B*** cell, which has passed into the state of a young epithelioid cell whose pedicle was at ***A,*** and which shows three arched acid-fast bacilli ***d, e, f,*** which are dumbbell-shaped chromatic rods. Other examples of the same kind will be found by examining the figure.

From the examination of these figures, the impression quickly emerges that the rods before our eyes are nothing other than the chromatic rods that we studied in p**lates 40 and 41** and that we saw so clearly in the embryonic cells ***a*** and ***d,*** **plate 40** and ***a,*** **plate 41.**

Pl. 42. *Section of tuberculous lung. Grosst 700. Agr. 1,250.*
Pl. 43. *Sections of tuberculous lung. Fig. 1. Gross 700. Agr. 1,250. Fig. 2 and 3. Gross 1,100. Agr. 2,000.*

We have also demonstrated, at this time, that the granulations of embryonic cells give rise to filaments which form the tissue of the trabeculae and that these filaments are constituted by dumbbells placed end to end or by a series of refracting balls, connected together by rods, and forming the image of a knotted rope. We have also seen that the chromatic dumbbell rods of embryonic cells stain strongly with acid fuchsin and are acid-fast, while the filaments formed by a series of dumbbells of the trabeculae of tuberculous tissue stain more weakly and are not acid-fast.

Thus, Koch's bacilli, whose presence we observe in the tuberculous tissue of lung sections, are chromatic rods of embryonic cells still contained in the mother cells or more or less dispersed when these are disintegrated at maturity. Also bacilli are the more or less colored and more or less acid-resistant dumbbell-shaped rods that we saw emerging from the granules by germination, to constitute the trabeculae of the tuberculous tissue. Koch's bacillus is therefore formed by embryonic cells, or rather, it is the element that constitutes them. It is the chromatic dumbbell-shaped rod of these cells or chromosome, and also the dumbbell-shaped germinative filament emitted by the granules of the adult cell.

This appears, at first glance, not to be in agreement with the classical morphological knowledge currently accepted on the tubercle bacillus. We will see later that this is not the case; the new observations, which will be presented later, will fully confirm the conclusion which has just been drawn.

Research into the origin of Koch's bacillus in the caseous matter of caverns.

The study which has been carried out here is that of the constitution of caseous matter. From the beginning of this research, facts of great importance have appeared.

I first studied the caseous matter of large caverns, and there, what was above all observed was the mode of formation of epithelioid cells by coalescence of several embryonic cells.

But as soon as I examined the caseous matter of the small cavities, about 3 to 5 millimeters in diameter, I noted the facts which are fixed by the photographs contained in plates 45, 46, 47, 48, 49, 50.

In **plate 45**, the reader will be able to note:

1. In figures 1, 2, 3, the presence of numerous typical Koch's bacilli, granular or not, most of them having the shape of dumbbells.

2. The formation of Koch's dumbbell bacilli by the granulations of embryonic or epithelioid cells. In Figure 1, the epithelioid cell or group of embryonic cells ***a,*** in the process of fusion, gives rise to the dumbbell bacilli ***b*** and ***c.*** In Figure 2, a completely disintegrated epithelioid cell shows the dumbbell bacilli, such as ***a, b, c,*** which form it exclusively; in the same figure, two confluent embryonic cells give rise to the non-granular bacillus ***d*** and to the bacillus ***e,*** which arises from a chromatic granulation /. Figure 4 shows an epithelioid cell ***a,*** not disorganized, but already old and not very colorable; it is formed exclusively of granular or non-granular dumbbell-shaped rods, such as ***b, c,*** and which are Koch's bacilli.

In Figure 3, a group of bacilli, ***e, j, k, l, m,*** arises from the granulations of the embryonic cell ***a;*** another group of bacilli, ***g, h, i,*** arises from the granulations of the embryonic cell ***b,*** then two others ***d, f,*** from different cells.

3. Some of the various forms and aspects that epithelioid cells take in caseous matter. We see very strongly colored chromatic granules as in cell ***b,*** figure 6, very refractile colorless granules as in cells ***b,*** figure 5 and ***a, b, c,*** figure 6.

4. In figure 7, the reader will see, taken in the act itself, one might say, the birth of bacilli and tufts of bacilli or bacillary filaments on the granules of the embryonic cells that have reached maturity; ***a*** is a large mature embryonic cell whose granules are germinating; they each emit a filament, which, almost immediately afterward, forms a new refractile or colored ball, thus constituting a dumbbell-shaped bacillus which will be extended by a new filament and a new ball, and so on.

Thus, this **plate 45** already establishes the formation of Koch's bacillus by the granulations of embryonic cells.

Plate 46 confirms this fact and provides new evidence so clear that the no doubt can remain. The caseous material examined was very rich in mature and germinating embryonic cells; these cells being examined successively and by and with varying focus, it can be seen that almost all of them emit bacilli. Some emitted a whole cluster of them.

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In Figure 1 of this **plate 46,** we see in a two bacilli of characteristic shape, granular, bearing on one side (the one which is well focused), their small refracting, uncolored ball; when a ball is not exactly in focus, we know that it gives a larger image whose outline has lost its sharpness; also the ball at the other end of the bacillus can only be guessed at by the white halo which surrounds this end.

Pl. 44. *Sections of tuberculous lung. Figs. 1, 2, 3, 4. Gross 1,100. Agr. 2,000.*
Pl.45. *Formation of Koch's bacillus by embryonic cells. Gross 1,100. Agr. 2,000.*
Pl.46. *Formation of Koch's bacillus by embryonic cells. Grosst 1.100. Agr. 2.000.*

We see in ***g,*** a group of five embryonic cells giving birth to characteristic bacilli, such as the granular bacillus ***d.***

In ***b*** (fig. 1), we see a group of three cells, two of which are disaggregated and show bacilli emerging on their granulations; we see there two large, strongly colored dumb-bell-shaped bacilli ***h*** and ***i*** and, next to them, other less colored bacilli, ***j, c,*** for example, equipped with their refracting balls and showing their characteristic dumbbell shape.

It will be apparent, by examining Figures 1 and 2 in detail (for example, ***a, c, e, f, i, j,*** then region ***l, m,*** Figure 1 and ***b, c, d, e, f, g, h, i, m, n, o, p, q, r,*** Figure 2), that the dumb-bell shape is the constant shape of Koch's bacillus. I indicate as the characteristic shape for the solid bacillus, the bacilli ***h, i, j, e, k, n,*** Figure 1, ***n, o, q,*** Figure 2 and for the granular bacilli, ***a, d,*** Figure 1, ***c,*** Figure 2.

The reader will be able, by searching in Figures 1 and 2, to see many other dumbbell-shaped bacilli. One will also be able to note the presence of bacilli in embryonic cells, such as ***p, q, r,*** in cell ***v*** and ***s, t, u,*** in cell ***x.***

In this figure 2, in addition to the bacilli that are born everywhere on the cells, we will see a whole group formed by the embryonic cell ***a.***

Let us note here that from the examination of these two figures, as well as those of the previous plates, the fact emerges that the bacilli, at the time of their formation, are large, uniformly and strongly colored, like bacilli ***h, i, n***, figure 1, ***c, f,*** figure 2; later, light points appear, as in bacillus ***o,*** figure 1, then the light zones increase in width, but the colored zones are still linear, that is to say longer than wide, as in bacilli ***a, e, p,*** figure 1; the colored points then decrease in length to constitute only small round granulations, three to six in number per bacillus, such as those of bacillus ***d,*** figure 1; these granulations also disappear and the bacillus is then a filament emptied of its chromatin and barely stainable such as ***r,*** figure 1, ***d, i,*** figure 2.

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We will add to these demonstrations those provided by figures 1 and 2 of **plate 47** and by **plate 48.** These three figures represent the caseous substance of the caverns of the lung. We take a little of this caseous substance, preferably from a small cavern, we put a particle like a pinhead in a drop of water placed on a glass slide and we gently agitate the caseous mass to dilute it in the water; we then spread the drop of water, fix it on the heating plate, then we color first with hemalun (hematoxylin) and then with Ziehl's fuchsin. In such preparations, here is what we see:

Pl.47. *Vegetation of Koch's bacillus in the caseous matter of the caverns. Gross. 1,100. Agr. 2,000.*
Pl. 48. *Vegetation of Koch's bacillus in the caseous matter of caves. Gross 1,100. Agr. 2,000.*

1. Strongly colored masses (***A, B,*** fig. 1, **plate 47**, ***A, B, C, D,*** fig. 2, **plate 47**, ***A, B, C, D,*** **plate 48**) consisting of chromatin-rich or achromatic granules and embryonic cells. From these masses arise tufts or bundles of bacilli, such as tuft ***C,*** figure 1, **plate 44,** and the length of these tufts shows that they are made up, not of isolated bacilli, but of long filaments which, at a given moment, will segment into bacilli. These tufts explain the presence of bundles of bacilli arranged in parallel in the sputum.

2. A considerable quantity of bacilli, some thick, rich in chromatin and strongly colored, others less colored, finally a large number which take on almost no color. Now it can be seen that, in this considerable number, and whatever the intensity of their coloration, all the bacilli are dumbbell-shaped and have a ball, generally refractile, at each of the two ends. We will see highly chromatic bacilli, for example, in ***i, n, f,*** figure 1 and ***b, e, f,*** fig. 2, **plate 47** and in ***c, d, j, o, q,*** **plate 48.**

As for the less colored dumbbell-shaped bacilli of characteristic shape, they exist everywhere, in all the regions of **plates 47 and 48.**

In Figure 1, **Plate 47**, we see at ***j, a, a,*** fairly long bacillary filaments which are made up of dumbbell-shaped elements placed end to end, their granulations being in contact. However, in certain places, we do not see two balls side by side, but only one with a rod at each end of the diameter, which represents the shape that I have designated as a knotted rope. We will examine filaments of this type again later.

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4. In the tuft of bacilli ***C,*** figure 1, **plate 47**, we notice that the bacilli form, at certain points along their path, a large, generally oval granulation ***a, b, c,*** larger than the intrabacillary granulations seen in ***p, b,*** figure 1, **plate 47**, for example; some of these granulations are simply one of the balls, loaded with chromatin, of a dumbbell-shaped element; these large oval granulations are eliminated from the body of the filaments, since at a given moment they are no longer seen there and, on the other hand, similar ones are found free in the vicinity.

* * *

The formation of long bacillary filaments, such as those in bundle ***C*** of Figure 1, **Plate 47**, is of great importance and it is important to study it specially; by searching in preparations of caseous matter, made as previously explained, one can quite easily find analogous filaments formed by the granulations of embryonic cells; **Plate 49** shows these filaments in all their stages of development. Figure 1 shows, enlarged in Figure 5, the birth of two bacillary filaments, ***c, f,*** clearly starting from two granulations forming part of the mass of two fused embryonic cells ***d,*** and ***e,*** which, in addition, give rise to other typical bacilli ***g, h, i.***

Filament ***c,*** Figure 5, gives us a demonstration that is useful to note; we saw in **Plate 48,** Figures 2, 3, 4, 5, that the mature granules of embryonic cells are rich in chromatin and stain very strongly; now, granulation ***a,*** Figure 5, **Plate 49,** which was rich in chromatin, has already lost it in half ***a*** and still has some in half ***b,*** from where the bacillary filament ***c*** starts, and it is visible that the chromatin that is missing at ***q*** is that which has entered the bacillary filament between points ***b*** and ***c.*** We will see the phenomenon fully accomplished in the bacillus ***k,*** figure 1, **plate 47**, one of whose balls ***l*** no longer has chromatin as in the bacillus, the ball ***m*** being the only one loaded with it.

In other bacilli, and this is the most frequent case, as we know, the chromatin contracts into small balls which constitute Much's granules; will these granules fuse in one of the balls at the ends of the bacilli, or are they expelled outside? They are surely expelled outside when the terminal balls are detached.

In figure 3, we see several short filaments leaving in a bundle from an embryonic cell. In figure 4, we see another bundle of three or four bacillary filaments leaving an embryonic cell.

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In Figure 6, a bacillary bundle of three or four filaments starts from an embryonic cell located at the very bottom of the figure and to the right. Another small similar bundle starts from an embryonic cell located below the center and a little to the left of the figure, of which only the silhouette can be seen. Note in this Figure 6 the birth of typical Koch's bacilli on the granules of an embryonic cell located a little above the center and a little to the left of the figure.

Figures 7 and 8, **Plate 49**, give us the complete demonstration we are looking for; they show that Koch's bacilli are segmentation elements of long filaments formed by embryonic cells. These two Figures 7 and 8 are reproduced enlarged in **Plate 50.** Here, the bacillary filaments visibly start from various embryonic cells. On one of the bundles in Figure 1, **Plate 50,** we will see the refracting balls which separate the rods ***a, b, c, d;*** the same remark can be made in the region ***k, l, m***, of the same figure 1. It can also be made in the region ***c, d, e, f,*** Figure 2, then on the short filament ***m, n, o, p, q,*** arising from the granulation ***m.***

According to **A.-Ch.** and **G. Hollande (1)** then **Kahn (2)**, imperceptible granulations are formed from Much's corpuscles by successive divisions, which are connected by fine filaments that are also barely perceptible.

(1) *A.-Ch. and G. Hollande, Soc. de Biologie, April 13, 1929.*
(2) *Kahn, American Review of Tuberculosis, August 1929; Annales de l'Inst. Pasteur, 1930.*
Pl. 49. *Germination of embryonic cell granulations and formation of long filaments in the shape of dumbbells or knotted ropes. - Fig. 5. -Magnification 1,100. Agr. 2,400. Figs. 1, 2, 3, 4, 6, 7, 8, Magnification 1,100.*

According to **Kahn**, the very fine granulations formed by division and of immeasurable size, gather in clusters and he adds: ''*These clusters remain unchanged for variable periods of time (twenty-four, forty-eight hours or more). However, one can see the most delicate rods imaginable being born at their periphery. At the beginning, these rods are so slender that they are difficult to identify. They grow with variable speed until the moment when they become adult acid-fast bacilli.*''

I have not sought to verify these facts because the mode of formation of Koch's bacillus by the chromatic granules of embryonic cells is established and photographed so many times in **plates 47 to 49** that there can be no doubt in this regard.

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The presence, in tuberculosis cultures, of large chromatic granules in large numbers and of the same dimensions as those of the embryonic cells, indicates that the element necessary for the multiplication of Koch's bacillus exists there.

The fact that the bacilli are placed end to end in cultures to form filaments as in tuberculous tissue shows that the mode of formation of the dumbbell-like elements must be the same in both cases.

We are now certain of the mode of formation of Koch's bacillus and of the place of this formation; in a word, we have seen it born and we know from what it is originally born: the chromatic granulation of an embryonic cell. We know that its constant form is the dumbbell shape, that is to say, a rod bearing a small ball at each end.

Now, this is not the form which is described and accepted for Koch's bacillus; it is described as a straight or arched, granular rod, having at its ends a width equal to that of the other parts.

This comes both from the fact that at a given moment, the terminal balls detach from the bacillus or that, still existing at its ends, they are so little visible, generally hardly staining, that they are overlooked; on the other hand, and because of the shape recognized in Koch's bacillus, the observer who sees one or two balls at its ends concludes that this is only a coincidence, an effect of chance which seems to be demonstrated by the presence of free balls in the field examined. Finally, the real existence of a certain number of bacilli not provided with terminal balls is explained by the existence, in bacillary filaments such as those in figures 6, 7, 8, **plate XLIX**, of fairly long portions devoid of balls and whose segmentation, not including the latter, would give rise to bacilli which do not possess them; this leads to the possibility of the existence of bacilli bearing only one ball.

To complete our knowledge of the nature and origin of Koch's bacillus, we must now enter into new considerations. The granules, which are, with the rods that unite them,

the exclusive elements of embryonic cells, constitute what is called the chondriome, each granule being considered a mitochondrion; **A.-Ch**. and **G. Hollande** consider the chondriome of the embryonic cell as a "primitive chondriome" from which the mitochondria of the plasma cells, which he calls primordial mitochondria, would derive by subdivision, these subdividing again to form the mitochondria of epithelioid and giant cells.

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The facts presented so far show that the granules or mitochondria of embryonic cells are not simple granules, but straight, arched, or **U**-shaped dumbbell rods, carrying the latter at their ends.

It will be demonstrated later, on page 135, that this dumbbell shape is the general shape of mitochondria, as much in a myelinated nerve fiber or a liver cell as in an embryonic cell. As we have previously demonstrated that the dumbbell rod is the fundamental element in the construction of anatomical elements, tissues, and the entire organism, we arrive at this double conclusion:

1. That it is the dumbbell mitochondrion that is the fundamental element in the construction of the organism.

2. That Koch's bacillus is the mitochondrion of the anatomical elements of tissues.

Let us now summarize the facts demonstrated in this chapter relating to the origin and nature of Koch's bacillus:

1. Koch's bacillus arises from the granules of embryonic cells, which themselves originate from the anatomical elements of the organism. It is, originally, the dumbbell-shaped mitochondria of these cells.

2. Koch's bacillus is therefore formed by the animal organism itself, that is, by the living matter that constitutes its organization.

3. It follows from these facts that the in vitro culture of Koch's bacillus is, in reality, a culture of the living matter of the organized being and, for greater precision, a culture of the fundamental living elements or mitochondria that serve to build the organism.

It is therefore necessary now to determine the forms and characteristics of the vegetation of Koch's bacillus in its in vitro cultures.

Constitution of Koch's bacillus cultures.

I studied cultures of human tuberculosis on glycerin-coated potatoes and in a veil on glycerin broth, which were provided to me by the Bacteriology Laboratory of the Faculty of Medicine, and other cultures on Lævenstein medium, which were provided to me by **Dr. Ameuille's** laboratory.

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The study was very simple; it consisted of taking plates of about half a square centimeter from the culture with a spatula, carefully placing them in a dish containing 5% formalin and then placing them in contact, without shaking, with 95° alcohol, then absolute alcohol, and finally xylol, and then embedding them in paraffin. The sections are then made flat, that is, in a direction parallel to the surface of the culture.

Placed on slides, they are then deparaffinized with xylol, then treated with hemalun and then with Ziehl's acid fuchsin with a small amount of alcohol added (1/4 to 1/3), this is mainly to avoid the deposition of fine fuchsin granulations on the sections.

The sections are examined thus stained and without passing them into an acid solution, our aim here being to study, not the acid resistance of the elements of the culture, but all the elements it contains.

The observations that I was able to make on preparations thus obtained relate to the following different points that we will examine successively:

1. Granulations and granular masses of the culture.

When we examine a section of a culture of human tuberculosis on glycerin-coated potatoes, made and prepared as indicated above, we are struck by the variety of aspects it presents.

In certain places, we see a multitude of characteristic Koch bacilli forming compact masses and scattered around them; in other places, we hardly notice any bacilli anymore and the section shows very varied figures which will be found photographed in the plates which follow.

These diverse aspects, according to the points considered, are due, obviously and above all, to the age of the elements.

We will examine, to begin with, a microscopic field photographed at a high magnification and represented in **plate 51,** with a total photographic enlargement of approximately 2000.

In this figure, we first distinguish a mass ***A, B,*** consisting of an intertwining of filaments and granulations, and clearly having the appearance of areolar tissue.

In this mass, we see a multitude of granulations of different sizes; the smallest, marked with a small cross in the ***D E F*** regions, are Much granules, that is, the fine granulations of Koch's bacilli.

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They are either freed from the bacilli, or contained in the bacillary bodies that have become colorless and almost invisible. These are chromatic granulations, but they do not all color with the same intensity. Those that take on the most intense color are the youngest.

Other much larger granules are, some strongly colored such as those marked with the letters, ***b*** at the top of the figure, others ***d,*** weakly colored; some uncolored and strongly refractive are manifested by a white spot, often bearing in the center or on the side, a small black point which is a Much granule which has come to stick to it. These large granules, colored or not, are of the same nature; they owe their coloring to the chromatin which they contain and become colorless when they have lost this, having passed into a bacillary filament, then into the opposite granulation terminating the bacillus. Indeed, we often notice that one of the balls of a dumbbell is colorless, the other colored. It has also been shown previously, regarding the origin of Koch's bacillus, that when a granulation forms a bacillary filament (**pl. 49**, fig. 5), the chromatin of this granulation gradually passes into the filament, there comes a time when the granulation, having lost its chromatin in one half, has retained it in the other from which the bacillary filament starts.

These large granules have a size of 0.3 to 1 m and even more; those marked with the letter ***d,*** for example, are about 1 m; those which end the dumbbell rods ***l, m, n, o,*** have 0.5 to 0.7 m on average.

These granules can come from two different origins. Some come from the fusion of several Much's granules; we see these granules fusing in groups of 2 to 4 or 5.

Other much larger colored masses, up to 2 to 3 µm in size, are visible in the figure at ***e, f, k.*** They can be seen and examined in all the figures in **plates 52, 53, and 54**. But they are especially easy to see in figure 2, **plate 52**; this is a point in the culture that was undergoing particularly active multiplication. There we see very large granulations such as ***a, c, d, n,*** and others of medium size such as ***h, j, e.***

Pl. 50*. Enlargement to 2,000 of figures 7, 8 of plate 49.*
Pl. 51. *Section of a culture of human tuberculosis. Agr. Gross. 2,000.*
Pl. 52. *Section of a culture of human tuberculosis on glycerin-coated potato. Gross 335. Agr. 600. Fig. 3. - Gross 335. Agr. 750. Figs. 1 and 2.*

On careful examination of the figure, we will realize that all the very distinct granulations are part of a dumbbell-shaped ele- ment, and we will be able to observe the presence of a good number of these dumbbells, very clearly visible, in many points of the figure.

The same observations relating to granulations and dumbbell-shaped elements can be made by examining figures 1 and 2, **plate 53**, and 1 and 2, plate 54.

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2. Bacillary elements and their relationship to granulations.

By examining the central region ***G,* plate 51**, we notice numerous dumbbell-shaped rods in ***l, m, n, o, p;*** the dumbbell arrangement is so clear that it can leave no doubt. If we examine very carefully and with a magnifying glass certain points, such as region ***l,*** of the same figure, we see that the dumbbell-shaped rods are in reality a filament interrupted by balls and give the appearance of a knotted rope; in ***l, l', l",*** for example, four rods separated by balls are placed end to end and appear to be part of the same filament; another dumbbell-shaped rod is, in addition, placed at each end.

The dumbbell shape of the rods or bacilli is not localized to this region; others are seen at other points and can be found in all points of the figure; they are very numerous in the ***z*** regions, where the tissue is much more compact, and where we realize that all the elements are dumbbell-shaped; it is the same in the other regions of the figure and the reader can make the same observation in all the figures of plates 51, 52, 53, 54.

He will be able to see them, in particular, in ***A, a, b, r, s, t, v, b, q, u, h, k, l,*** fig. 1, and ***k, l,*** fig. 2, **plate 52**; in ***d, f, n,*** figure 1, **plate 53** and in ***B, W, m', r, q, s, c,*** figure 1, **plate 54,** and he will find many of them by looking for them himself with a weak magnifying glass.

In vitro tuberculosis cultures are therefore made up, like tuberculous tissue, of dumbbell-shaped elements; this shape is therefore the normal shape of Koch's bacillus.

3. Grouping of the knotted rope filaments and the dumbbell-shaped rods into bundles and anastomosis of these.

The knotted rope filaments and the dumbbell rods are grouped into small bundles such as ***s, q, r,*** which, anastomosing with others, form large cavities such as ***v, x, y,*** (**pl. 51**). Note, on the other hand, that we see in ***G,*** by the filaments ***l, m, n, o,*** the elementary process which forms the trabeculae of the areolar framework described previously and will determine, by multiplication, the filling of the cavities ***G, x,*** etc. (**pl. 51**), with tuberculous tissue.

Pl. 53. *Section of a culture of human tuberculosis on glycerin-coated potato. Figs. 1 and 2. t Gross 335. Agr. 600.*

The areolar tissue formed by these small anastomosing bundles will be seen in extremely demonstrative forms in Figure 3, **Plate 52**, in ***a,*** Figure 1 and in ***c, d, e, f,*** then ***o, g, n***, and in ***j,*** Figure 2, **Plate 53;** finally everywhere in Figures 1 and 2 of **Plate 54.**

This areolar tissue with large meshes is identical to that which was previously described in connection with the forms of tuberculous tissue and exposed in several plates.

As for the finer areolar or reticular tissue which constitutes the internal mass of the giant cells, its equivalent will be found in most of the figures; but it will be found with complete identity in the regions ***h, i, f, j, c,*** figure 1, **plate 53** and between the points ***A, g, h, s, v,*** of figure 1, **plate 54;** to convince oneself of this identity, one will make the comparison with the reticular tissue of the two giant cells of plates 21 and 22.

The two figures on **Plate 53** show a fact that is observed identically in pulmonary lesions. Developing tuberculous tissue is always, at first, areolar and lacunar; as it develops, the lacunae become smaller and gradually fill in at the end. Generally, at least, the lacunae are completely filled, and the tissue has become completely compact when it reaches the caseous state. In Figure 1, **Plate 53,** we see tissue in ***h*** that has become compact, while in ***a,*** the younger tissue is still lacunar.

In Figure 2, it is part ***b, c, d, o, n, p, i*** that is the younger and, as it develops, forms extensions of areolar tissue that advance as they develop, while part ***a, m, k,*** the older, has already become compact.

5. Formation of figures having the shape of epithelial cells with their framework.

For example, in Figure 2, **Plate 53,** at ***j,*** we will find a formation resembling one of the epithelial cells in the process of disorganization, such as those seen in **Plates 14 and 19,** with the frame at ***t, h,*** and rays coming from it to converge towards the center. At ***d,*** Fig. 1, **Plate 52,** we will see a central mass from which start of numerous divergent rays and which has the appearance of a nucleus formed by them.

Pl. 54. *Section of a culture of human tuberculosis on glycerin potato. Figs. 1 and 2. Magn. 335. Agr. 600.*

6. Tendency towards the formation, in cultures, of a framework similar to the supporting framework of the inter-alveolar septum and to the formation of bundles similar to those which constitute the frameworks of epithelial cells.

The facts that I report here are most curious, because they show how persistent, in organic elements transported in in vitro culture, are the organizational properties they possess and their ability to take on specific forms conducive to the creation of organized tissues and organs.

Plate 55 contains photographs of four different fields of a section of a human tuberculosis culture on Loevenstein medium, about thirty-five days old. In these four figures, we will notice the same sinuous, irregular appearance of the bundles which, by their anastomosis, constitute, as in cultures made on different media, an areolar framework having certain points of resemblance to the framework supporting the inter-alveolar septum. We see numerous points where the bundles have formed a cell similar to those in the inter-alveolar septum which contain the epithelial cells. We will see two of these cells at the top right of Figure 1, then throughout Figure 4.

As on the supporting framework of the inter-alveolar septum, we notice the formation of small pyriform masses, some of which are pedunculated, having exactly the shape and dimensions of embryonic cells. We will see them in figures 1 and 2, **plate 55.**

As for the rods in dumbbell-shaped bacilli, they exist everywhere in this **plate 55**, but the magnification (about 335) is too low to be able to see them without looking for them with a strong magnifying glass.

The figures in this plate will be usefully compared to those in plates 7, 8 and 9, relating to the inter-alveolar septum.

Plate 56 represents the photograph of a veil of a culture of human tuberculosis on glycerin broth, about three weeks old. The nutrient medium of the culture being different from that of the cultures examined previously, its appearance naturally changes, but the essential characteristics of the culture persist.

The general appearance of the veil is represented, at two different points, by Figures 2 and 3, at a magnification of about 50. A point from Figure 2 was photographed in Figure 1 at a magnification of 335. There we see anastomosing bundles of different sizes constituting areolas and giving exactly the image of the intermediate matrix or gangue of the tuberculous tissue.

Pl. 55. *Section of a culture of human tuberculosis on Loewenstein medium.*

In Figure 4, several isolated bundles are photographed on the edge of the veil, in the process of development, showing their tortuous, divaricate appearance and the birth of the lateral branches they emit. Note that they form, in the center of the figure, ovoid bodies which are identical to embryonic cells; we will frequently see some of an identical elongated ovoid shape in several previous plates representing sections of pulmonary lesions.

* * *

This study of the constitution of human tuberculosis cultures allows us to conclude:

1. That there is complete identity between the tissue formed by an in vitro tuberculosis culture and the tuberculous tissue of pulmonary lesions; the same identity is observed between their constituent elements.

In both cases, the same areolar tissue is formed, the essential constituent elements of which are the dumbbell rod and its granulations.

2. That Koch's bacillus exists in in vitro tuberculosis cultures in the same dumbbell rod form as in the elements of tuberculous tissue and in the normal anatomical elements of the lung.

3. That, since it has been proven that tuberculous tissue is made up of the same fundamental elements as those of the normal anatomical elements of pulmonary tissue, it becomes evident that tuberculosis cultures are an in vitro culture of the fundamental elements of tissue, the dumbbell mitochondria; the properties preserved by these elements in the cultures confirm this origin.

It is, in short, an in vitro culture of elements of lung tissue which, although having retained a certain power of organization, are incapable of forming complete epithelial cells and capillary vessels.

I recall that, in the first volume of this work, I concluded that tuberculosis is an autogenous disease and that Koch's bacillus was formed by the organism itself. ***This is therefore correct.***

But I had also concluded that Koch's bacillus resulted from a modification or transformation of the normal organic mold. ***This conclusion was therefore inaccurate.***

Koch's bacillus is the mitochondria or normal constituent element of anatomical elements. If this mitochondria vegetates in an abnormal way to constitute tubercular tissue, it is not because it has changed shape, but because the conditions of the environment in which it lives have changed.

(1) *Gunther Enderlein, The Biological Reasons for the Success of Sanitary Living Conditions in Combating Tuberculosis; The Tubercle Bacillus, a Developmental Stage of Aspergillus, Mold, Arch. f. Ententwicklungsgeschichte derbakterien, 1931, p. 6.*

From a long study in which he compared the development of Koch's bacillus with that of molds of the genus Aspergillus, ***G. Enderlein (1)*** *concluded that Koch's bacillus is a stage in the development of the different species of molds of the genus Aspergillus.* ***The conclusion he drew is therefore inaccurate.***

In the first volume of the work "*Constitution of Animal and Plant Organisms*..., etc." published in 1926, I demonstrated that tuberculosis cultures can be transformed quite easily in vitro into molds of various forms, notably into the *Mucor* and *Aspergillus* forms.

I outlined the attempts I had made to induce tuberculosis by inoculation of the various forms of mold that I had obtained, either by culturing fragments of tuberculous lung or by transforming cultures of Koch's bacillus. I have already explained why I abandoned this method of research for the study of the nature and origin of tuberculosis.

It was undoubtedly the transformation of tuberculosis cultures into molds of the genus *Aspergillus,* which I published in 1926, that prompted **Enderlein** to research the relationships between Koch's bacillus and *Aspergillus.*

His conclusion that Koch's bacillus is a stage in the development of Aspergillus ***is not correct,*** firstly, because a culture of Koch's bacillus can be transformed into *Penicilium* and *Mucor* just as easily as into *Aspergillus.* One might therefore just as well conclude that Koch's bacillus is a stage in the development of molds of the genera *Mucor* and *Penicilium.*

But the demonstration of the inaccuracy of **Enderlein's** conclusion results even more from the various facts that were observed during the study that is the subject of this work, and which together provide the demonstration of the origin and nature of Koch's bacillus, the normal mitochondria of anatomical elements.

SIGNIFICANCE OF THE FACTS OBSERVED
CAUSE AND NATURE OF TUBERCULOSIS

The new knowledge provided by the studies that have just been presented is based exclusively on material facts recorded by photography, and shows us that the development of pulmonary tuberculosis occurs in humans as follows:

1. Initially, there is an inflammatory or congestive, pneumonic lesion of the lung tissue, of a non-tuberculous nature. Such a lesion affects the pulmonary epithelium and, at certain points, the capillary vessels. This fact is known and does not need to be demonstrated again.

2. Any lesion of this kind has a tendency to repair and heal itself; with a view to this repair, the inter-alveolar septum forms, by a true budding, young cells intended for the restoration of the epithelial cells; this fact is proven by plates 7, 8, 9; these are the ele-

ments that have been called embryonic cells. They therefore do not come from the blood by diapedesis and are in no way lymphatic cells.

3. When these cells do not fulfil the role of repairing the epithelial wall for which they were formed, they nevertheless continue their evolution, a fact proven by the formation, by some of them, of cells so similar to epithelial cells that they have been called epithelioid cells (a known and photographed fact, pl. 38, fig. 1). But most of these embryonic cells evolve in another way: the interior granules that they have formed by multiplication give rise to dumbbell-shaped elements which, by multiplication, constitute long filaments composed of a series of these elements placed end to end and emitting laterally ramifications of the same constitution. This germination of the granulations of the embryonic cells and this constitution of the dumbbell-shaped elements and dumbbell filaments is proven many times by the plates in this work.

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4. These dumbbell filaments unite to form bundles that anastomose and are the trabeculae of the intermediate framework encompassing all the cellular elements of the tuberculous tissue.

5. The dumbbell filaments united in bundles in turn emit a considerable quantity of new embryonic cells which evolve like the previous ones and come to constitute the entire mass of the tuberculous tissue, in the form of infiltration tissue and tuberculous follicles.

6. The dumbbell filaments, often very long, emitted by the embryonic cells (plates 49 and 50) will insinuate themselves far into the inter-alveolar partitions and are the agents of infiltration of the tuberculous tissue and the extension of the lesions.

7. The rods and dumbbell filaments which constitute the entire mass of the trabeculae and the tuberculous tissue gradually lose their chromatic substance and at the same time their acid resistance and then finally disappear, leaving only amorphous matter in their place. This is how caseous matter is formed, which is not necrotic tubercular tissue, but rather the tissue that has reached the end of its development. As all the elements are not at the same stage of their development, the caseous matter contains, at the same time, mixed amorphous matter, various granulations, dumbbell-shaped elements in various states, embryonic cells, and epithelioid cells.

8. The trabeculae of tubercular tissue, having fully developed, end up emptying their contents, either into the areolae they form, or into the cavities of the nearby air ducts, themselves altered. Thus, the caverns are formed.

The evolution of the tuberculous lesion is thus completely known and established, demonstrated by precise facts. The agent of the formation of tuberculous tissue is the

embryonic cell, and the agent of infiltration, that is to say, of the extension of the tuberculous tissue and the progressive destruction of neighboring healthy tissue, is the dumbbell-shaped germinative filament of the embryonic cell, a filament which has the property of itself forming new embryonic cells.

This is the certain process of the development of tuberculous lesions, established by precise, photographed material facts.

We followed this process to the terminal point of the lesion, the cavern, and up to this point, we did not have to take into account the influence that Koch's bacillus can exert on its development; and this fact was able to occur because no bacilli were seen in the tuberculous lesions that we studied.

To establish the relationships that exist between Koch's bacillus and tuberculous lesions, we had to seek special materials for this study.

* * *

If Koch's bacilli are not found in tuberculous lesions, even in the most characteristic, it is because they are sought in a form that is not the one they normally possess, and because they are attributed characteristics that they do not always possess.

Thus, by looking for it in the form of an isolated, granular, straight rod, 3 to 5 m long, it will obviously not be possible to detect it in trabeculae composed exclusively of dumbbell filaments 20 to 50 m long or more. It will be even less possible to detect it if, for this purpose, it is characterized by acid resistance, these dumbbell filaments becoming discolored by contact with the acid solutions used.

The presence of Koch's bacillus, with its currently accepted characteristics, is especially recognized when it is formed directly, either inside or outside an embryonic cell; this formation takes place especially in already old tuberculous tissue and even more so in the caseous matter of caverns rich in embryonic cells. And even in this case, Koch's bacillus is, as previously shown, generally provided with one or two of its terminal balls.

In the tuberculous tissue in formation, we have seen that the embryonic cells give rise to long, non-acid-fast filaments formed of dumbbells placed end to end and which constitute the trabeculae; these filaments do not segment on the spot. These are the reasons why the presence of Koch's bacillus is not often observed in tuberculous lesions when it is sought by current methods.

However, it is always present in every tuberculous lesion, since it is the fundamental and exclusive element that constitutes tuberculous tissue.

These observations necessarily lead us to investigate why this fundamental element, the dumbbell rod, which, at least morphologically, is the same in normal anatomical elements and in tuberculous tissue, multiplies abnormally to constitute the latter while it remains in relative rest in the anatomical elements.

The identification of the identity between Koch's dumbbell bacillus and the dumbbell rods or mitochondria of normal anatomical elements is quickly seen by examining these elements in plates 46, 49 to 54 on the one hand, and 14 to 19 on the other.

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The problem that lies at the origin of the research I have undertaken since 1920 is the following: *What are the physiological modifications that are the cause of the abnormal evolution, at a given moment, of anatomical elements or their constituent elements that have remained normal until then?*

The most important factor that regulates the organization and normal evolution of tissues is the normal circulation of blood plasma through the anatomical elements.

The alteration of this fundamental condition alone provides the desired explanation; as soon as blood circulation, and consequently that of blood plasma, is suppressed in the anatomical elements, their living conditions are completely changed; they are placed in the same conditions as anatomical elements detached from the tissues and deprived of circulation by trauma. The organization of these anatomical elements degenerates and dies, but their fundamental constituent elements remain alive; there may be repair; but if this is delayed, the fundamental elements of the degenerated parts, remaining alive, multiply freely, outside the laws of the organization of anatomical elements. In the same way, the influence of trauma and the causes of decay of anatomical elements (irritation, ulceration) on the development of cancer is explained.

In the development of tuberculosis, it appears that it is the evolution of embryonic cells and in particular the multiplication of their mitochondria, outside of any blood circulation, which is the cause of the unregulated and indefinite vegetation or proliferation of the latter, as in a mucor culture, for example, where the development of these same elements is limited only by the extent of the culture medium.

The preceding considerations having been set out, and the constant, obligatory, and exclusive presence of the dumbbell-shaped mitochondria **(1)** in all tuberculous lesions having been established, let us determine what role Koch's bacillus plays in their development.

(1) *I do not say Koch's bacillus because the latter, as defined, only responds to a particular, incomplete state of the actual constituent element of tuberculous tissue, the dumbbell-shaped mitochondria.*

Firstly, the facts we have set out allow us to affirm that it is not the primary causative agent of tuberculosis. *This primary causative agent is the embryonic cell born from the normal elements of pulmonary tissue.*

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The direct, immediate cause of tuberculous lesions is the abnormal development of embryonic cells which, instead of fulfilling their role of restoring the destroyed epithelium, vegetate and form long filaments, which constitute the tuberculous tissue and which insinuate themselves into the surrounding healthy tissues (infiltration) to multiply there. *This cause is, in summary, a disordered, limitless vegetation of the mitochondria of embryonic cells substituted for their normal vegetation, directed, regulated, and normally limited to the creation of an epithelial cell.*

Koch's bacillus therefore does not act as a causal agent of tuberculosis. The causal agent of tuberculosis is the embryonic cell, the agent producing all of the tuberculous tissue. The active agent of infiltration and extension of the lesions is the germinal filament of the chromatic granules of the embryonic cells, a filament consisting of a series of dumbbell-shaped rods or mitochondria.

Koch's bacillus, the product of the segmentation of these dumbbell-shaped filaments, is therefore only a witness to the evolution of tuberculous lesions.

If we consider Koch's bacillus with the characteristics that bacteriologists currently attribute to it—a granular rod of uniform diameter and acid-fast—we can only observe its presence in an irregular manner in the tuberculous tissue.

If we consider it with its real characteristics, with knowledge of its origin and nature, and of the differences it presents during its evolution, we see then that it is always present in every tuberculous lesion, since it is the exclusive formative agent of tuberculous tissue, being the primordial mitochondrion, the dumbbell-shaped element originating from embryonic cells, and even going back further, originating from the normal mitochondria of the anatomical elements of pulmonary tissue. Its real characteristics must make it defined:

A dumbbell-shaped element formed by a rod of variable size, also of variable length from 1 to 5 or 6 microns, provided at each end with a spherical granulation having 2 to 3 times the diameter of the rod, staining strongly and uniformly with Ziehl fuchsin when it is rich in chromatin (and acid-fast in this case), staining only very little when it has lost its chromatin, a loss which coincides with that of acid-fastness, taking on a granular appearance when it contains only a small quantity of chromatin in the form of Much's granules.

It may lose its two terminal balls, which gives it the appearance of a uniform rod. In lesions, the dumbbell-shaped elements are arranged end to end to form filaments and bundles of filaments.

Considered with these characteristics, the dumbbell-shaped element, of which the classical Koch bacillus is only an incomplete form, can be recognized and identified everywhere, at every point of a tuberculous lesion.

The facts presented so far, which provide us with knowledge of the nature and origin of tuberculous tissue and Koch's bacillus, constitute, at the same time, the demonstration of two facts of capital importance.

The first is that tuberculosis develops spontaneously in individuals, the tissue-destructive element being created by the organism itself and arising from the abnormal evolution of its normal constituent elements.

The second is the formation of an autogenous virus in a living organism by its normal constituent elements, and the mechanism of this formation; the first complete demonstration of this fact is given by the origin of Koch's bacillus.

The first of these facts, the spontaneous development of tuberculosis, leads to the necessary conclusion that contagion is not the initial cause of this development.

The second has a scope that is not restricted to the particular case of tuberculosis; it provides knowledge of the original source of viruses; they all necessarily come from living organisms.

The cause of the development of tuberculosis and the formation of tuberculous tissue is the same as that of the development of cancer and the formation of cancerous tissue; in the latter, embryonic cells are formed in the same way, arising from the mitochondria of normal tissue elements, and from trabeculae of cancerous tissue forming new embryonic cells whose evolution and role in the formation of cancerous tissue are the same as those of embryonic cells in tuberculous tissue.

The nature, origin, and elementary constitution of cancerous tissue will be described in a new volume currently in preparation.

THE DEVELOPMENT OF TUBERCULOSIS IS SPONTANEOUS.

ITS INITIAL CAUSE IS NOT CONTAGION, AS A RULE.

We now have knowledge of new, precise notions of material facts that give us the solution to this question.

In the numerous preceding observations, the results of which are fixed and recorded by photography, we have, so to speak, witnessed the development of tuberculous lesions.

We have seen the embryonic cell form by budding on the elements of the inter-alveolar septum, then we have seen it develop, becoming a sac of granulations which, by germinating, form the trabeculae of the tuberculous tissue, at the same time as new embryonic cells of a second generation, then of several successive generations.

We have seen that it is this newly formed tissue that is the tuberculous tissue, and that this, as it ages, becomes the caseous matter whose evacuation, through the airways, will form the cavities. We have finally witnessed the birth of Koch's bacillus formed by the granules of embryonic cells, elements formed by the alveolar wall, that is to say by the normal tissue of the lung to restore the destroyed epithelium.

Thus, by multiple pieces of evidence, all converging, the fact is established that *tuberculosis develops spontaneously in the individual and not by contagion.*

The sequence of facts formally demonstrates that it is not Koch's bacillus which, originally, is the cause of the development of the tuberculous lesion. The initial lesion is a lesion of the alveolar epithelium, an inflammatory or pneumonic lesion; *but the immediate cause of the development of the tuberculous lesion is the formation of epithelial cells, intended to repair the epithelium* and whose growth will form the tuberculous tissue.

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Koch's bacillus, the result and not the cause of the growth of these cells, is therefore not the cause of tuberculosis. The true cause of tuberculosis is, as previously explained, *the disordered, limitless growth and multiplication of the elements of embryonic cells, substituted for their normal growth and multiplication limited to the creation of an epithelial cell.*

So, in fact, the development of tuberculosis is spontaneous and contagion does not intervene, at least practically, to cause it.

It is now, and once this fact is established, that certain arguments or facts invoked to prove the non-existence or, at least, the great rarity of contagion take on their value; these are the facts opposed to **Villemin** by clinicians of the middle of the last century, by **Pidoux (1),** for example. These facts, notably the impossibility of providing direct,

manifest proof of contagion in humans, even in the case where the infection is daily, continuous, that of the spouse of a tubercular patient, for example, now have their full significance and really signify that contagion does not intervene, for its part, to cause cases of tuberculosis not due to spontaneous development, at least in adults.

Auguste Lumière (2) took up these arguments and analyzed, developed, and completed them in such a way that they constituted an imposing body of presumptions, if not proofs, opposed to the doctrine of contagiousness and of a nature to cast doubt on the accuracy of this doctrine.

His efforts were in vain because, it must be recognized, the transmission of tuberculosis by inoculation, discovered by **Villemin**, and the subsequent discovery of Koch's bacillus constituted, in the current state of knowledge, a position too strong to be shaken.

However, it can now be affirmed that there was, in **Villemin's** conclusions, an error of reasoning which consisted in not strictly limiting his conclusion to the scope of the observed fact: his experiments meant only that tuberculous matter, inoculated into animals, confers tuberculosis on them and that consequently they contain a tubercular virus.

They did not mean that this infection, caused experimentally and by inoculation, could occur under very different conditions, simply through contagion.

The results of the experiments of my late and illustrious master **A. Chauveau,** on the contamination of calves by ingestion of tuberculous matter through the digestive tract, which reinforced **Villemin's** conclusions, only proved the possibility of human contamination in the particular case of ingestion of massive doses of tuberculous matter; they did not prove the existence of this contagion under very different conditions.

Currently, the new facts presented in this work demonstrate the spontaneous development of tuberculosis; the clinical facts opposed by clinicians of the last century and even by current clinicians to the doctrine of contagion, take on, alongside the preceding facts, the value of positive facts intended not only to demonstrate the non-intervention of contagion, but also to shed light on the causes of the selectivity of the spontaneous evolution of tuberculosis in certain individuals and not in others.

But, the elements of the question being thus specified, it is impossible to affirm that, in exceptional cases, contagion cannot occur. No fact invalidates this possibility; on the contrary, certain facts tend to demonstrate it, such as the contamination of calves by the

(1) *Bull. of the Association of Medecines, 1867 and 1868.*
(2) *A. Lumière, The Medical World, No. 865, June 1935; The Health Movement, No. 140, December 1935.*

digestive route in **Chauveau's** experiments, the contamination of guinea pigs by the normal air route, using dried tubercular sputum, in **G. Küss's** experiments... etc.

I do not wish to dwell further on this question of the possibility of contagion, because it could only be fruitfully addressed by providing new, precise facts and, on the other hand, because being rare and exceptional, if it exists, it is of only secondary importance, the spontaneous evolution of tuberculosis having been demonstrated.

I will not discuss further the question of infantile contagion, on which I have no new documents; but nevertheless, I think that the facts I have presented obviously do not apply only to adult humans and that it is quite probable that the development of tuberculosis is also spontaneous in children.

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ORIGINAL SOURCE OF VIRUSES

The research presented in the first volume of this work led me to conclude, following the study of the elementary constitution of the tissues of animal and plant organisms:

1. That all viruses come from animal and plant organisms, living or dead, and that they cannot come from any other source.

2. That, in any organism, an autogenous virus can be born by modification of what I called the organic mold characteristic of the species.

In this first volume, I explained the process by which I sought to find the original source of heterogeneous viruses. I demonstrated, following a long series of research, that the constituent elements of tissues cultivated in vitro remain alive, continue to vegetate, and that this vegetation manifests itself in the form of a mycelial culture, that is, a mold culture, and also in the form of bacterial cultures.

By the processes I have indicated, I had transformed into mold cultures the pure bacterial cultures that are the viruses of known diseases; I had photographed all the forms of these molds; then, on the other hand, I had transformed the living substance of the muscles, liver, spleen, etc., of numerous animals, as well as the living substance of numerous plants into various forms of their specific mold. I then compared the mold forms of the viruses with those of living animal and plant tissues. I thus hoped to deduce, from the identity I would observe, from which living organism, animal or plant, a virus came.

I add that I was justified in making this comparison because I had previously made the anatomical demonstration that organic tissues are all made up of elements identical to molds.

A first result demonstrated to me that the principle I was using was correct. Having studied extensively the organic mold of the potato and its transformation forms, I was perfectly familiar with its Peronospora form, which I had isolated; studying the transformations of Trichophyton Cratériforme, I also succeeded in obtaining its Peronospora form, which was identical to that of the potato. Immediately, inoculation tests were attempted on the rabbit's skin, shaved and slightly scarified, with the various molds from the potato. Most determined a characteristic trichophytosis. This result encouraged me to continue my research using the same method. I did indeed obtain the identities of the forms I was looking for and deduced the original source of the viruses. I thus concluded on a simple resemblance and without providing any material proof.

I thought that I could, once this first stage of determination was completed, prove experimentally and by other methods the accuracy of my conclusions.

I devoted several years to research in this direction without obtaining the desired result. I then focused my research on the study of smallpox, having in this regard two easy means of checking the accuracy of my results: obtaining the vaccine pustule and creating immunity against vaccine inoculation. I obtained interesting results in this study which will be published in a future volume, but I have not yet succeeded in determining the original source of the vaccine.

This entire series of research demonstrated to me that I was on the wrong track and that, although the basis of the research was accurate, it was insufficient to lead to a favorable result.

I thought at that moment that there must probably be, in the constitution and mode of formation of viruses, important facts that I was unaware of and that needed to be determined in order to successfully pursue the search for their original source.

It seemed to me that research into the mode of formation of the tuberculosis virus was entirely appropriate for this study, since the form of the virus was known and I had already determined that the original source of the human tuberculosis virus is the human organism.

This is the origin of the research presented in this second volume. Let us now examine the result from the special point of view that concerns us here.

We have reached the point where we see Koch's bacillus being born, that is, the point where we see what it is born from and in what form. It is born from a chromatic granulation of the nucleus of a cell, the embryonic cell, and in its characteristic dumbbell shape.

By this fact, material proof is therefore provided:

1. That the original source of the human Koch bacillus, a virus capable of tuberculizing other species by inoculation, is the human organism.

2. That the original source of viruses is the organism of living beings.

Thus, here is established, for the first time, the original source of a virus and the mode of its formation in the animal organism.

It is quite obvious that this is not a special case and that the demonstration made applies to all viruses, autogenous or heterogeneous.

A virus must be called a substance which, introduced into the animal organism by an abnormal route, multiplies its elements represented in the bacterial form and causes pathological phenomena there.

Viruses are all made up of the living matter of animal and plant organisms, and these are also our food. The ingestion of the latter through the digestive tract and the penetration of the products of their digestion through the intestinal mucosa must necessarily take place in the form of a chemically transformed matter so divided that it cannot, by fusion of elementary particles, reproduce the original living matter of the food with its specific characteristics. If the food is incompletely transformed and if the intestinal penetration routes are in an abnormal state, it is admissible that these two facts together could be the causes of an infection through the intestinal route.

But since viruses are all made up of living matter, it must be admitted that they are all destroyed by digestion in a normal digestive tract. This fact appears to be so consistent with reality that I have been led to repeat, under various conditions, the experiments that established the contagion of tuberculosis through the ingestion of tuberculous matter. These experiments are currently being carried out.

It appears that, if the tuberculosis virus were not constantly destroyed in the digestive tract, all tuberculosis patients who swallowed their sputum rich in tuberculosis material would die very quickly from generalized tuberculosis because, if the tuberculosis virus crossed the intestinal mucosa in viral form, it would be disseminated everywhere via the lymphatic and then blood routes.

The mitochondria of tissues are dumbbell-shaped elements, of which some histologists have seen only the granulations at the ends, and some others only the rod that connects them.

We have seen that these are the granulations of embryonic cells, that is, the primordial mitochondria of these cells that form the dumbbell-shaped elements that we have identified, without any possible doubt, with Koch's bacillus. The fact that the form of the mitochondria is perpetuated without morphological change in cultures of Koch's bacillus shows that these are indeed a culture of mitochondria, that is, fundamental elements of tissues, which means that, in reality, Koch's bacillus does not exist in the state of a characterized species; it is only a special form or state of mitochondria, fundamental elements of the organism.

Since the fundamental elements of the organism of animal species have specific characteristics particular to each species, we understand why cultures of human, bovine, or avian tuberculosis, which are cultures of mitochondria from humans, cattle, or birds, have distinct characteristics that allow them to be recognized.

Koch's bacillus is therefore not, as has been believed until now, a pathogenic bacterial species that would live and reproduce who knows where, probably in the organisms it infects, and whose existence would be preserved by passing from one organism to another.

Since a culture emulsion of Koch's bacillus causes, when injected into the veins, the development of lesions in an animal's organs, it is indeed a virus. But this notion must be supplemented by adding that it is the living matter of the animal from which the culture originally originated that is, in reality, the virus. The proof is easy to provide: an emulsion of grey matter from the cerebral hemispheres of an animal, injected into the veins, also causes the development of pulmonary, hepatic, and other lesions. Therefore, the grey matter of the cerebral hemispheres of animals constitutes a virus.

The serum of an animal, rich in living figurative elements, notably mitochondria or mitochondrial elements, is a virus and it is in this capacity that it causes serum sickness. These facts are not new; the principle has already been explained in the first volume of this work.

They provide knowledge of the original source of viruses: the organism of all organized beings, animals and plants, is the original source of all viruses, and they can emit this virus into the external environment, either during life or after organic death by disintegration (decomposition of bodies) of the constituent elements. These facts show the need to study human diseases with new guiding ideas.

Most human diseases are accompanied by more or less significant changes in the tissues, in different parts of the organism, and these modifications can be accompanied by the appearance of various bacterial forms, the cause of which is an abnormal, pathological evolution of the fundamental elements of the organism. It is certain that a large number of these bacterial forms are autogenous. It is all the more necessary to study them and not to consider them as coming from external infections, since certain bacillary forms are normal and the rule in the evolution of the organism; it was demonstrated, in the first volume of this work, that the existence of these forms is normal in the blood of animals.

The facts presented also provide new and crucial knowledge on other points that we will examine successively.

1. On the formation of bacterial cultures and hyphomycete cultures by the living matter of the organism of animals and plants.

2. On the elementary constitution of living and organized organic matter.

Formation of bacterial cultures and hyphomycete cultures by the living matter of the organism of animals and plants.

I must, once again, repeat that we have witnessed, in my demonstrations, the formation of Koch's bacillus, by the chromatic granulation of embryonic cells.

This is the demonstration, fixed in all its stages by photography, of the transformation of a chromatic granulation, belonging to a cell, into one or more bacilli, that is to say, of the creation of a bacterial culture by the living matter (the chromatic, nuclear substance, for greater precision) of the animal organism.

I had clearly observed this fact of the creation of bacterial cultures by living matter and had explained it at length in the first volume of this work; I had seen the transformation of animal or plant tissue, cultivated in vitro, into mycelial or bacterial culture and I had demonstrated, by multiple proofs, that one can easily transform a mycelial culture into a bacterial culture and vice versa.

The publication of these facts, however simple, in the first volume of this work, was received with a skepticism that was to be expected, due to the opposition they present with currently accepted principles.

The fact, stated in the first volume of this work, that a piece of liver or lung can give rise to a bacterial culture was considered absurd. I was offered aseptic guinea pigs, from

which I could not obtain any culture; I am still waiting for these animals, and this certainly because the accuracy of my observations has been established. It has been claimed, and the objection was foreseen, that the animals used in my experiments were infected.

The answer to these criticisms is now given by the demonstration of the creation of Koch's bacillus by the chromatic granules of an embryonic cell born from lung tissue.

It will also be possible to prove that urinary colibacillosis, for example, is of renal origin and comes from the creation of coli bacilli by the epithelial cells of the kidney, because coli bacillus is, obviously, another autogenous virus that can form in various points of the body.

As for the direct formation of molds by tissue cultured in vitro, proof of the accuracy of this fact is extremely easy to provide directly; in fact, the culture of Koch's bacillus, culture of tissue mitochondria, is transformed, without great difficulty, into molds of the forms *Penicilium, Aspergillus, Mucor*, etc.

This fact also made many scholars smile; a bacillus which transforms into *Penicilium*, a bacteria which transforms, not only into another species, nor even another genus, but into a living species of another class...!

Now, the facts are beginning to receive their explanation. We will see later that the genus *Mucor* is made up of already organized living matter. *A mycelial filament of mucor is made up of dumbbell-shaped mitochondria and organized almost exactly, by these mitochondria, like the myelin nerve fibers of animals.*

In summary, current research fully confirms, by demonstrating the origin of the Koch bacillus and the mechanism of its formation, the following two fundamental principles established in the previous volume of this work:

1. The living matter of the tissues of organized beings is capable of transforming into bacterial culture.

2. The original source of pathogenic viruses is the organism of animals and plants. The human body is the original source of Koch's bacillus.

CONSEQUENCE OF THE FACTS FROM THE POINT OF VIEW OF THE DIAGNOSIS OF TUBERCULOSIS.

Knowledge of the nature and origin of Koch's bacillus, an element derived from the normal mitochondria of tissues, gives us an explanation for certain phenomena such as the frequency of positive reactions to tuberculin, so great in humans that several observers have observed it in 98% of humans chosen at random.

The morphological identity between the elements of tuberculosis cultures and the fundamental, normal elements of tissues, the mitochondria, and, on the other hand, the fact that the former are born from the latter, bring to the question new knowledge which completely modifies the significance of the reaction to tuberculin. It is understood that, under these conditions, all healthy, non-tuberculous subjects react; it is also evident that, under these conditions, a positive reaction is not proof of the existence of a tuberculous lesion, and that *the existence of the reaction alone can have no value from the point of view of the diagnosis of tuberculosis.*

As for the intensity of the reaction, it is possible that it is related to the presence of a tuberculous lesion; this is a question that cannot be discussed here. The question is of great economic importance if examined from the point of view of the regulations imposed on bovines reacting to tuberculin. I will not discuss this question here either; but I nevertheless draw attention to the absolute necessity of revising it. The new facts brought forward in this work concerning the origin and nature of tuberculosis, the origin and nature of Koch's bacillus, provide all the elements necessary for a rapid solution to this question.

Let us note, in this regard, that the presence of a few bacilli identical to the tuberculous bacillus in the abdominal lymph nodes of an animal, for example, is not sufficient to affirm, as has been done, the existence of a tuberculous infection.

If we refer to the normal shapes of tissue elements and compare them with those of the elements of tuberculosis cultures, we will see that it is impossible, by morphological appearance alone, to discern whether it is one or the other.

We have seen previously that, in all tuberculous lesions, there is one element that is never missing, even if we do not see characteristic acid-fast bacilli. These are the embryonic cells. Since these cells are easily recognizable, easy to distinguish from epithelial cells, and moreover they give rise to Koch's bacilli, it is obvious that they constitute a more reliable sign of tuberculosis than the presence of the latter.

Moreover, the embryonic cells form well before Koch's bacilli, thus, allowing a much earlier diagnosis.

However, attention must be drawn to the fact that after an acute, non-tuberculous inflammatory lesion of the lung tissue, there is a phenomenon of repair of the epithelial elements which must take place by means of embryonic cells formed by budding. It is therefore possible that at the end of pneumonia, for example, there is expulsion of embryonic cells through the sputum; in this case, and if this elimination is only temporary, it is obvious that it cannot be interpreted as a sign of tuberculosis. There are therefore conditions here which must be established by a clinical and microscopic study.

Since the embryonic cell is the direct causal agent of tuberculosis, it is therefore this cell which, in the sputum, constitutes the earliest and most reliable element in the diagnosis of tuberculosis, and which can allow this diagnosis in the absence of bacilli.

These facts and those preceding them also explain how inoculating a guinea pig with sputum containing anatomical elements of the alveolar walls can prevent the development of tuberculous lesions and thus cause a serious error in diagnosis.

It has indeed been proven in several plates that the anatomical elements of the alveolar wall, notably the epithelial cells (pl. 12, for example), and their debris, can give rise to embryonic cells. However, it has also been demonstrated that it is these that give rise to Koch's bacillus.

The method of diagnosing tuberculosis by inoculating guinea pigs with sputum and, more generally, with material containing anatomical elements or their debris, must therefore be rejected. Diagnosis by microscopic study of sputum must take into account the actual shape of Koch's bacillus, the dumbbell shape, and the fact that this dumbbell-shaped Koch's bacillus has very often lost its acid resistance.

Under these conditions, the diagnosis becomes difficult to establish, because dumbbell-shaped bacillary forms, and even simple rod-shaped ones that have lost the two balls of the dumbbell, can be eliminated from the lungs with the sputum in any pulmonary disease. This occurs particularly whenever epithelial cells are eliminated in the sputum, and this explains why Koch's bacilli, which are said to be non-virulent, can be found in the nasal cavities or pharynx of healthy individuals. This shows that the diagnosis of tuberculosis by Koch's bacillus can give rise to errors and that, in difficult cases, it is the presence of embryonic cells, and especially their persistent proliferation, combined with their germination, showing the formation of bacilli at their periphery, which will be the reliable, error-free diagnostic element.

Consequences of the facts observed from the point of view of the treatment of tuberculosis.

The facts we have observed teach us that tuberculous tissue is formed by elements identical to the normal elements of the organism and that Koch's bacillus is, in short, only a special aspect that these elements take on, the proof of which is provided by the fact that, in an in vitro culture of Koch's bacillus, the fundamental elements are identical to those of the tissues.

In short, tuberculous tissue is formed from normal elements of the organism whose evolution and organization are no longer directed, or which have lost their aptitude for organization. We do not know whether this loss is real in the elements or whether it corresponds only to the loss or insufficiency of something else, the blood supply, for example; but it is very likely that this factor plays a capital role.

Under these conditions, it is no longer surprising that 98% of individuals react to tuberculin. This means that all humans react, and it means, at the same time, that it is not because they are tubercular that they react, but because tuberculin, derived from an in vitro culture of fundamental tissue elements, influences their normal fundamental elements.

The origin, nature, and constitution of tuberculous tissues and in vitro tuberculosis cultures leads us to the conclusion that wanting to achieve immunity against the action of Koch's bacillus is an error, since it is not the immediate cause of the development of tuberculosis; this immediate cause is the embryonic cell and the vegetation of its mitochondria.

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It is therefore against this vegetation, against the elements that it produces that should be immunized; now, these elements being identical to those of the organism, it follows that the problem would consist, ultimately, in finding a means which would have the effect of destroying or stopping the development of the elements of normal tissues as well as those of tuberculous tissue.

Thus, the demonstration of the immediate cause of the development of tuberculosis, its origin and its nature, leads to these simple, elementary notions:

1. That one cannot immunize against this disease because it is not a disease of heterogeneous bacterial cause, but a form of abnormal evolution of tissues. This does not mean that one cannot even definitively stop the evolution of this disease by other means.

Therefore, any research aimed at obtaining an immunizing culture or serum is a chimera; moreover, if no serum, no body, no culture can achieve immunity against tuberculosis, one can affirm, on the other hand, the certainty of succeeding in achieving, in a certain number of cases, by treatments of this nature, the infection of the subject or the development of a serum sickness.

It is therefore impossible, due to the nature and origin of tuberculosis, that treatment with BCG, in any form whatsoever, can have an immunizing action.

2. The various chemical treatments directed against Koch's bacillus cannot have any favorable results for the same reason. A product that would destroy tuberculous tissue will destroy normal lung tissue even more quickly.

3. Since the immediate cause of tuberculosis is an abnormal development, a disruption of the vegetation of embryonic cells, it is this vegetation that the treatment must aim to restore to its normal form.

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ELEMENTARY CONSTITUTION OF LIVING AND ORGANIZED MATTER

To determine whether the development of tuberculosis is spontaneous, that is, whether Koch's bacillus arises spontaneously in the body, I had to, in order to discover how it arises, research what relationships the bacilli have with the tissues at the points where their presence is observed.

The first observation made was that certain tubercle bacilli, isolated or in groups of three or four, carry at their end a barely colored refractile ball or seem to start from this ball, while others carry a ball at each end.

Next, I observed that when the bacilli are in groups of three or four, they are born on a group of granules and that, sometimes, these granules are still coherent enough to show that they were part of the same disintegrating embryonic cell. Finally, we can see identical dumbbell shapes clearly included in embryonic cells that are still complete and even have their pedicles. These shapes were described on pages 90 to 93 and I indicated at that time that they must be considered as chromosomes.

Dumbbell bacilli also form, born externally on the peripheral granules of embryonic cells. It is easily seen that these dumbbell shapes, whether isolated, in groups, or included in cells, are identical and have the same origin. Some have the characteristic appearance of Koch's bacillus with its Much granules.

I had noted, at the same time, that numerous discolored, that is, non-acid-resistant, dumbbell-shaped elements were contained in the large trabeculae of the tuberculous tissue. I had already determined, previously, that these trabeculae are made up of fila-

ments resulting from the germination of embryonic cells, and that they contain, in addition, numerous refractile granulations; but I had not noticed the dumbbell-shaped elements there.

-135-

There was therefore a connection between the dumbbell-shaped elements formed by the granulations of the cells, and those observed everywhere in the trabeculae. Observation led me to see that the filaments of the trabeculae are, in reality, dumbbell-shaped elements placed end to end, or a filament carrying, at close spaces, refracting granulations; when a trabeculae emits a branch, it is in the form of one or more dumbbell - shaped elements which branch off from it and of which one ball is in direct contact with a ball of a dumbbell of the trabeculae.

I also saw that the pedicle of the embryonic cells is made up of one or two filaments placed side by side; often, and in this case it has the shape of a club, it is made up of a single dumbbell-shaped element, one of the granulations of which is included or placed at the edge of the embryonic cell.

It follows from these facts that the trabeculae are made up of a mass of dumbbell - shaped elements, affecting various arrangements, and originating from the vegetation and multiplication of the first dumbbell-shaped elements formed by the granulations of the embryonic cells.

The first elements formed are acid-resistant, the oldest are no longer so. It is, in short, a tissue, the tuberculous tissue, which the dumbbell-shaped elements form.

It became curious to investigate whether the body's normal tissues contain these same elements. I looked for them, first, in the epithelial cells detached from the alveolar wall or still adherent, in the trabeculae that constitute the framework of the inter-alveolar septum, in the vessel walls and their epithelial cells. I found them everywhere and noted that they are the only constituent element of the organization, with granulations that appear isolated, but which can also belong to dumbbell-shaped rods when viewed end-on.

To understand the constitution of epithelial cells, refer to plates 14, 15, 16, and 17. We have seen, regarding the description of these cells (page 49), that they are generally constituted by a frame or envelope from which concentric rays depart, connecting the nucleus to the periphery. It is the peripheral frame visible in ***g', k',*** **plate 14**, in ***a, d, f,*** figure 1 and ***b,*** figure 2, **plate 15,** which allows us to reliably differentiate the epithelial cell from the epithelioid cell, the latter not having a frame.

Frame ***b,*** figure 2, **plate 15,** is constituted by dumbbell-shaped elements, as is the rest of the cell.

Examination of cells ***g, h, i*** shows that the radiating filaments which connect the frame to the nucleus are constituted exclusively by dumbbell-shaped elements. Examination of cell ***g',*** **plate 14,** clearly shows dumbbell-shaped elements. Examination of cell g', plate 14, also clearly shows, by the few remaining radiating filaments, o, o, o, that they are made up of dumbbell-shaped elements; the same observation will be made on the met n cells of the same plate.

A similar observation can be made by examining the epithelial cells of plates 16 and 17.

As for the part of the inter-alveolar septum that I called the supporting carcass, and which is visible in **plate 14**, at ***B, f, i, h"***, it is very apparent, particularly at points ***f*** and ***E,*** that it is made up of dumbbell-shaped elements.

By looking in normal rabbit tissues, liver tissue, cerebral white and grey matter, myelinated nerve fiber, I observed the same constitution of the anatomical elements by dumbbell-shaped elements.

Thus, the anatomical elements of the organism are made up of dumbbell-shaped rods and not the simple granules that were considered to constitute the mitochondrial apparatus, that is, the mitochondria, elements whose role was until now only hypothetical. Current observations specify that this important and unknown role of the mitochondria is to be the constructive element of the tissues and of the entire organism; this role is analogous to that played, in a finished house, by the brick and rubble whose assembly constitutes the walls.

* * *

I also wanted to determine whether the dumbbell shape is indeed the completely general constructive element in all living beings; for this, I turned to those placed at the very bottom of the scale of animal and plant organisms, to molds.

In the first volume of this work, published in 1926, I explained and demonstrated that the constitution of the mycelial filaments of molds of the genus *Mucor* is identical to that of myelinated nerve fibers. For this demonstration, I used various species of *Mucor,* including *Mucor Regneri,* prepared by impregnation with silver nitrate.

Figure 3 of **Plate 57** has already been included (Fig. 5) in **Plate 50** of the work cited above; but, at that time, the rods dumbbell-shaped had escaped my attention.

Plate 56. *Horizontal section of the veil of a three-week-old culture of human tuberculosis on glycerin broth. - Fig. 1. Magn. 335. Fig. 2, 3. Magn. 60. - Fig. 4. Magn. 550.*

I took new photographs of the same preparation from which this figure came, and enlarged them. Figures 1 and 2 of **plate 57** represent two of these photographs.

As for figure 3, it is an enlargement of figure 4, **plate 50**, of the work cited above; it represents a myelinated fiber from the rabbit sciatica dissociated and impregnated with silver nitrate.

A quick examination of all the points designated by numbers in Figures 1, 2, and 3 shows that the *Mucor* filaments, which consist of a cylindrical axis and an envelope or sheath, like myelin fibers, are made up exclusively of dumbbell-shaped elements identical in shape and size to those that constitute all tuberculous tissue, giant and embryonic cells, and epithelial cells and other elements of the inter-alveolar septa of the lung. Many of these dumbbells are so distinct that the slightest doubt can remain as to their complete identity with those that constitute animal tissues, normal or pathological.

On careful examination of the figures, one will see not only dumbbell-shaped rods, but the dumbbell filaments or knotted rope filaments that I have described in tuberculous tissue.

We will see some of varying length in 12, 13, 14, 22, 23, 24, 25, 26, 27, - 15, 17, 18, 19, filament ***B*** figure 1, **plate 57.** We will see a long one in ***q, q, q, q,*** at the lower part of filament ***E*** and the reader will be able to see others by looking with a magnifying glass in the figures.

As for the dumbbells, we will see some very clear ones for example in 1, 3, 4, 6, 7, filament ***A;*** the letter ***l*** shows only one in the center of a group of three dumbbells placed side by side. The letter ***l*** filament ***E*** also shows one in the middle of a group of three.

Another remarkable arrangement, identical to that which exists in myelinated nerve fibers, is visible at the external part of the axis cylinder of filament ***A;*** dumbbell-shaped fibrils detach from it and come in an oblique direction, spreading out in the external sheath which they themselves constitute, since we can see (fig. 2) that this is constituted exclusively by dumbbells. This arrangement is very clear in the region of the numbers 7, 8, 9, 10, 11; dumbbell 11, for example, terminated by ball 10, is continued by two other rods separated by ball 9; the terminal ball touches the external membrane.

Pl. 57. *Figs. 1 to 4. Mycelial filaments of Mucor Regneri. Gross. 335. Agr. 600.- Fig. 5. Myelinated nerve fiber of the rabbit sciatic nerve. Impregnation with silver nitrate. Gross. 600. Agr. 1,500.*

This arrangement is also apparent in figure 3, **Plate 47**; dumbbells such as 3 detach from the axial cylinder to join the external membrane; dumbbell 10 detaches almost perpendicularly from the axial cylinder to join the periphery, and it is in contact with lateral dumbbells which seem to detach from it.

Figure 3 represents mycelial filaments that are almost empty and of which only the external membrane is visible; very clear dumbbell shapes can be seen at 2, 3, and 4, and the composition of the external sheath can be seen in the vicinity of these points.

Figure 4 represents a myelin fiber from the rabbit sciatic nerve, dissociated and impregnated with silver nitrate. Upon careful examination, the same observations can be made as in the *Mucor* filaments. Dumbbells can be seen at all the points marked with letters; for example, there are four at point 14, and others at 10, 11, 12, 13, 15, 16, and 17.

From the axis cylinder, dumbbells such as 1, 2, 3, 4, 5, extend toward the periphery; for dumbbells 4 and 5, for example, the terminal ball is in contact with the balls of two diverging dumbbells 6, 7, 8, 9, which end at the Schwann sheath or are in contact, like dumbbell 7, with others that go there. These are the branched tracts or rods that **Nageotte** described as connecting the axis cylinder to the Schwann sheath and which will be discussed later.

Plate 47 therefore shows us, with great clarity:

1. That the central part of the *Mucor* filaments, identical to the axial cylinder of myelin nerve fibers, is composed, like the latter, of fibrils and that these are, in reality, dumbbell or knotted rope filaments. The small ramifications that leave this central part, to form the outer envelope of the filament, are constituted, at their start, by a dumbbell-shaped element with a granulation placed on the edge of the axial cylinder; the same arrangement exists in myelin fibers;

2. That the filaments of molds of the genus *Mucor* are constituted by dumbbell-shaped elements identical to those that constitute normal or pathological animal tissues, that is to say, by mitochondria.

This is, of course, only a morphological identity that does not extend to the chemical constitution or the properties inherent to these elements in each organ.

Jean Nageotte, who extensively studied the structure of the myelin sheath in his remarkable work entitled "*The Organization of Matter*," **(1)** described:

(1) *J. Nageotte. "The Organization of Matter in its Relations with Life," Paris, 1982. Alcan.*

1. In a nerve treated with acetic bichromate and ferric hematoxylin, fine tracts divided dichotomously, which cross the entire thickness of the myelin sheath to attach themselves to its external limit (p. 227). He adds that "*there is continuity between the substance of the axial cylinder and that of these tracts*."

2. An enormous quantity of granular filaments oblique in various directions, which appear in place of the branched tracts, when the preparation is stained by the Altman method. These filaments, which resemble bacilli, are very clearly individualized. They terminate freely at each of their ends; they sit in the branched tracts, but they remain simple. And **Nageotte** adds: "*these filaments are mitochondria, there is no doubt about it, and the tracts, in which they are placed, are in continuity with the neurite.*"

After various considerations, **Nageotte** adds that the conclusion he has arrived at is that *the myelin sheath is, in all likelihood, a gigantic compound mitochondrion.*

This conclusion shows us that **Nageotte** has managed to observe and partially conceive of the organization of the myelin sheath, although the dumbbell element remained unknown to him.

In **Nageotte's** description, it is easy to guess that the granular rods, which go from the axis cylinder to the periphery of the nerve fiber, are the dumbbell-shaped elements whose two refracting balls were not visible in his preparations, one being attached to the axis cylinder, the other being at the periphery or attached to a ball of a second dumbbell-shaped element which heads towards the periphery.

I will now quickly complete the new observations that I was able to make relating to the myelin sheath and which are absolutely identical for the external sheath of the mucor filaments.

The external terminal ball of the dumbbell-shaped elements which connect the axis cylinder to the external membrane of the myelin sheath is part of this membrane and is related to a ball of another dumbbell-shaped element which, this one, is located in the plane of the curved surface of the membrane which it constitutes by its connection and its entanglement with other similar elements.

It follows, therefore, that the radiating branches, constituted by dumbbell-shaped elements *which start from the axial cylinder, have the clearly marked function of going to the peripheral membrane of the fiber to constitute it, and to maintain its cylindrical shape.* If, on the other hand, we consider that the axial cylinder is not made up of simple fibrils, but also of dumbbell-shaped elements, and that the radiating branches that emanate from it are directly related to them and are their extension, it appears that it is

not only the myelin sheath, but the entire myelin fiber, which could be likened to a gigantic mitochondrion, following **Nageotte's** expression; in fact, the myelin nerve fiber is, in its entirety, a tissue formed by dumbbell-shaped mitochondria or knotted rope filaments.

As we have seen that an epithelial cell of the lung, an embryonic cell and the tuberculous tissue it forms, a liver cell, a mucor filament, are constituted exclusively by this dumbbell-shaped mitochondrion, it is therefore not only the myelin fiber that would be a gigantic mitochondrion, but the entire organism, because the fundamental element of its construction is the dumbbell-shaped rod.

It follows from this fact, absolutely general for all organized beings, that the word mitochondrion does not designate anything in particular. Created to designate granules or rods to which a role of the utmost importance and moreover unknown was attributed, it turns out that the element thus designated is the fundamental element of the construction of tissues.

Of this fundamental dumbbell-shaped element, some have seen only the balls at the ends, others only the filament joining the two balls, like, for example, the rods described by **Nageotte** in the myelin sheath of nerve fibers.

Thus is outlined the study of the elementary constitution of living organisms and the processes that achieve the organization of living matter, as well as the complete construction of an organism.

Of this study, I have only wanted to expose the elementary principles here, without developing them further, because this question will be, along with the study of the nature and origin of cancer, the subject of the third volume of this work, currently in preparation.

The facts that have just been presented in this work bring such profound modifications to some of the current knowledge, notably to that concerning the nature and origin of tuberculosis, its contagiousness, the nature and origin of Koch's bacillus, that they necessarily call for a check. To carry this out, it is sufficient to examine some microscopic preparations of tuberculous human lung, and some preparations of cultures of human tuberculosis on glycerinated potato, established as I have indicated, by the method of sections after inclusion in paraffin.

Such an examination will make it possible to successively verify the accuracy of the following facts:

1. Embryonic cells are born by a pedicle, a dumbbell-shaped element, either on a branch, or on the alveolar wall, or on a desquamated or non-desquamated epithelial cell.

2. The embryonic cells germinate; from them come dumbbell-shaped elements that constitute the framework of the tuberculous tissue or the reticular tissue of giant and epithelioid cells.

3. The embryonic cells form, as they develop, granulations that give rise to the dumbbell -shaped elements that constitute the framework of the tuberculous tissue and are at the same time Koch's bacillus.

4. Cultures of Koch's bacillus are formed by the same dumbbell-shaped elements that originate from the embryonic cells.

5. These dumbbell-shaped elements are those which constitute the framework or framework of the epithelial cells and other elements of the inter-alveolar septum; the terminal granules or balls of these elements are those which have been called mitochondria, and which constitute the chondritis or mitochondrial apparatus; the mitochondria are dumbbell-shaped elements, constituted by two granules or balls, connected by a rod of a smaller width; the length of this rod is very variable, it can even be less long than the diameter of a ball or reach four to six times this diameter or even more.

Verifying these facts is all the easier since they are all recorded by multiple photographs that precisely reproduce the images of the objects I have described and in the very setting in which I observed them.

In summary, the problem is very simple and is contained entirely in this fact:

The entire evolution of tuberculosis merges with that of the embryonic cell; this cell is born from a mitochondrion of the normal elements of the alveolar wall, and it forms, in turn, new mitochondria that will constitute the tuberculous tissue and Koch's bacillus.

Let us recall that the importance of control is considerably increased by the fact that a second problem is solved at the same time as the previous one: knowledge of the original source of viruses and the creation of autogenous bacterial viruses by the animal organism. The solution of this second problem is at least as important as the first for the progress of studies relating to human diseases.

END

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TRANSLATED EXPLANATORY TABLE OF PLATES
(for Volume I)

(1) *The magnifications indicated for the drawings are approximate.*

Plate 1. Formation of germinal masses (leukocytes) in an in vitro bacterial culture (Tetanus transformation mold, reduced to the bacterial state). FIG. 1, gr. 610: Photograph of the elements of the original culture. FIG. 2, Agr. (gr. 1220).

Pl. 2. FIG. 1, gr. 1100: Elements of a syphilis bacterial culture on agar on the eleventh day(drawing). FIG. 2, gr. 1100: Elements of a one-month anaerobic tetanus broth culture (drawing). FIG. 3, gr. 1100: Spore-forming hyphae in a gonococcus culture on agar on the twenty-sixth day (drawing). FIG. 4, gr. 1100 **(1)**: Mycelial elements and germinative masses of a diphtheria culture on agar on the fortieth day (drawing).

Pl. 3. Elements of a tubercular sputum culture: FIG. 1, gr. 1100, in broth on the seventh day (drawing). FIG. 2, gr. 1100, in broth on the twenty-eighth day (elements of the surface veil (drawing). - FIG. 3, gr. 1100, on agar on the seventh day (drawing).

Pl. 4. FIG. 1, gr. 245, left part; gr. 1100, right part 2: Germinal masses of a typhus bacillus culture on agar on the fourth day (drawing). FIG. 2, gr. 1100: Germinal mass of a bacterial culture on agar from the liver of a normal guinea pig. - FIG. 3: Elements of an aerobic tetanus culture on gelatin after fifteen hours, twenty-three hours, and thirty hours (Gr. 1100).

Pl. 5. Gr. 1100**:** Elements of a tetanus culture in the rat pleura (pleural fluid after twenty-six hours) (drawing).

Pl. 6. FIG. 1**:** Formation of heterogeneous leukocytes in the cerebrospinal fluid of the dog twenty-four hours after injection of a bacterial culture of the Penicilium form of the organic coffee mold. Photograph of the elements of the cerebrospinal fluid. Enlargement; gr. 1100×2=2200). - FIG. 2: Formation of identical heterogeneous leukocytes in vitro bacterial broth culture. Photograph of the culture elements (Enlargement; gr. 1100×2=2200).

Pl. 7. FIG. 1, gr. 1100**:** Original photograph of Figure 1, Plate 6. FIG. 2, gr. 1100: Another aspect of the same preparation.

Pl. 8. Formation of germinative masses or heterogeneous leukocytes twenty-four hours after injection, into the cerebrospinal fluid of the dog, of spores of a mold of the type Mucor racemosus. Various aspects of a preparation of cerebrospinal fluid (FIG. 1, gr. 350. FIG. 2, gr. 450). FIG. 3, gr. 690: 1, germinative mass in vegetation; 2, mycelial filaments that it emits; 3, fructifications that terminate the filaments.

Pl. 9. Vegetative elements of the sleeping sickness pathogen in the venous blood of the ear of a guinea pig inoculated with the Sporothricum form of the pathogenic mold. FIG. 2, gr. 100: Overall photograph of a group of two or three germ masses and the bundles of mycelial filaments they emit. FIG. 1, gr. 245: A, Group of two or three germ masses; B, C, the bundles of mycelial filaments they emit; D, mycelial filaments of the bundles; E, mycelial dilations that constitute the trypanosomes. FIG. 3, gr. 245: Formation of trypanosomes, F, by dilation of the ends of the mycelial filaments from the germ masses.

Pl. 10. - Vegetation of germ masses in in vitro bacterial cultures or in animal body fluids. FIG. 1, gr. 1100: Germ masses of a broth anthrax culture developing its mycelial filament. FIG. 2, gr. 1100: 1, 2, Germ masses of the sleeping sickness pathogen in the venous blood of a guinea pig inoculated with spores of the Sporothricum form of the pathogen; 1, a germ mass forming an almost complete trypanosome 3, and another 4, still rudimentary. FIG. 3, gr. 245: Vegetation of germ masses in the cerebrospinal fluid of a guinea pig inoculated with spores of a mold derived from a tetanus culture. FIC 4, gr. 245: Vegetation of germinal masses in a pneumonia patient's sputum. FIG. 5, gr. 1100: Development of germinal masses in breast cancer (drawing).

Pl. 11. Gr. 1100**:** Vegetative and fruiting elements of molds in the spleen of a normal gray mouse (drawing).

Pl. 12. FIG. 1, 2, 3, 4, gr. 245**:** Photographs of the vegetative and fruiting elements of molds in the spleen of a normal gray mouse.

Pl. 13. FIG. 1, 2, 3, gr. 245. FIG. 4, gr. 110**0:** Photographs of the vegetative and fruiting elements of molds in the spleen of the normal rat.

Pl. 14. Mold elements in the white matter of the hemispheres (FIG. 1, gr. 1100) and in the choroid plexuses of the normal rat (FIG. 2, gr. 1100). Drawings.

Pl. 15. - Mold elements in the white matter of the brain (FIG. 1, gr. 1100), in the spleen (FIG. 2, gr. 1100) of the normal cat, and in the spleen of the normal rat (FIG. 3, gr. 1100) (Drawings).

Pl. 16. - Mold elements in the white matter of the optic bodies (FIG. 1, gr. 1100), in the gray matter of the hemispheres (FIG. 2. 2, gr. 245. 1, 3, 4, 5, 6, 7, 8, gr. 1100), in the white matter of the hemispheres (FIG. 3, gr. 1100) of the normal pore and in the white matter of the hemispheres of an eight-day-old kid (FIG. 4, gr. 1100) (Drawings).

Pl. 17. Mold elements in the liver (FIG. 1, gr. 600), and the white matter of the hemispheres (FIG. 2, gr. 1100) of the normal ox (Drawings).

Pl.18. Mold elements in the liver of a normal ox (FIG. 1, gr. 610. FIG. 2, gr. 245), and in the spleen of a newborn rat (FIG. 3, gr. 245) (Photographs).

Pl. 19. Mold elements in the choroid plexuses (FIG. 1), and in the white matter of the hemispheres (FIG. 2), of a dog with rabies.

FIG. 1, gr. 1100**:** Mycelial trunk (3), its varicose ramifications (5, 6), and the spore-bearing hyphae (7) it forms. Varicose mycelial branch (1) bearing encapsulated balls (2) with stalks.

FIG. 2: 1, 2, gr. 610**:** Varicose mycelial branches ending in balls (11); 3 gr. 1100 Mycelial filaments ending in ball-shaped fruiting bodies; 4, idem; gr. 1100; 5 to 12, gr. 1100; 5: Lateral chlamydospores on a mycelial filament; 6: Germinative masses appearing to belong to the rabies mold; 7, 8, 9: Various mold formations; 10: Torn germinative mass letting its contents escape; in the center, bacillary form of the rabies agent.

Pl. 20. FIG. 1 to 4, gr. 525. - FIG. 5, gr. 610**:** Vegetative and fruiting elements of molds in the spleen of a dog injected with anti-spleen goat serum. Various aspects of a preparation.

Pl. 21. FIG. 1 to 5, gr. 245**:** Rudimentary spindle-and pear-shaped fruiting forms in a rudimentary culture of the Penicilium form of the human organic mold isolated from uterine cancer. FIG. 6, gr. 245: Mycelial development of anthrax on thin-layer broth. Ball-shaped fruiting bodies. FIG. 7, gr. 245: Ball-shaped fruiting bodies, later transforming into groups of sterigmata, of the Penicilium form isolated from the cerebrospinal fluid of patients with encephalitis lethargica.

Pl. 22. Intermuscular lamellar connective tissue of beef (intergluteal). FIG. 1, gr. 1100 approximately: Diagram of the elements of connective tissue (Drawing). FIG. 2, gr. 245: Fixed cells and their mycelial branches. -FIG. 3, gr. 245: Germinal masses in formation (Mastzellen). FIG. 4, gr. 610: Mold fruiting bodies in terminal balls. FIG. 5: Basophilic germinal masses of irregular shapes.

Pl. 23. Intermuscular lamellar connective tissue of the ox. -FIG. 1, gr. 610; 1, small germinative mass; 2, formation of large germinative masses or sclerotia by grouping and fusion of small masses. FIG. 2, gr. 245: Germinative masses. FIG. 3, gr. 100: Overall view of a preparation; some sclerotia are indicated by a cross. FIG. 4, gr. 100: A large sclerotium and the connective tissue bundles it emits. FIG. 5, gr. 245: The same sclerotium and the bundles it emits; the photograph is underdeveloped to make the sclerotium and its branches very visible.

Pl. 24. Lamellar connective tissue of the ox (Figs. 1 to 4, gr. 245). - FIG. 1: Large connective (mycelial) bundle, a lateral branch, its branches and their division into elementary fibers. FIG. 2: A mycelial branch, the lateral fibers and the fruiting bodies it emits.

FIG. 3**:** Flat cells of the bundles, mycelial dilations of elementary fibers. FIG. 4: The same flat cells on dissociated fibers.

Pl. 25. FIG. 1 to 5, gr. 245**:** Mycelial dilations on branches of the Mucor racemosus form of the human organic mold.

Pl. 26. Lamellar connective tissue of beef. FIG. 1 to 6, gr. 610: Various forms of fixed cells (mycelial dilations or germinative masses) of connective tissue and the mycelial branches they emit.

Pl. 27. FIG. 1, gr. 885**:** Germinative masses A, B, C, D, E, of a culture of Eberth's bacillus on agar, the branched mycelial filaments (cilia) they emit and the zymospores developed on them. FIG. 2, gr. 610: Development of the germinative masses, A, and dilated mycelial filaments B, of a mold derived from a tetanus culture. FIG. 3, gr. 245: Lamellar connective tissue of beef. Vegetation of the small germinative masses B, C, D, E, F, G, H. Formation of spores and zymospores on the mycelial filaments. -FIG. 4, gr. 610: Mycelial vegetative growth of the tetanus agent. Form FIG. 5, gr. 245: Mycelial vegetative growth of the scarlet fever agent.

Pl. 28. Lamellar connective tissue of the ox. FIG. 1, gr. 245: Fibrillar constitution of the mycelial branches. FIG. 2, gr. 245: Spore-bearing appendages of the connective fiber branches.

Pl. 29. - Lamellar connective tissue of the ox (Fig. 1, 3, 5, 6, 8, gr. 245. FIG. 4, gr. 100). Connective tissue of the periphery of a breast cancer (FIG. 2, 7, gr. 245). -FIG. 1: Constitution of the connective bundles. FIG. 3: Fruiting bodies of the connective fibers. -FIG. 4: Germinal masses (sclerotia) of the connective tissue. FIG. 5, 6: Formation of fruiting bodies by winding of the fibers. -FIG. 7: Fruiting body of Mucor racemosus on connective branches of the pericancerous area of a breast tumor. -FIG. 8: Fruiting bodies of the Monosterigmata form of connective fibers.

Pl. 30. Lamellar connective tissue of the ox. Formation of germinative masses; sclerotia (FIG. 1 to 8, gr. 245).

Pl. 31. Lamellar connective tissue of the ox (FIG. 4, gr. 245. - FIG. 1, 2, 5, 6, gr. 610. FIG. 3, gr. 1440). Spiral winding of connective tissue bundles and elementary fibers.

Pl. 31a. Formation of fat in connective tissue (FIG. 1, gr. 91). FIG. 2, gr. 245: Formation of fat cells by branched lateral branches of the connective tissue bundles. FIG. 3, 4, gr. 245: Sclerotia or fixed fat cells of connective tissue.

P1. 32. Vegetative forms of canine organic mold in vitro. - FIG. 1, gr. 610: Mold form obtained by culture and transformation of subcutaneous connective tissue. FIG. 2, gr. 245: Sterigmatocystis form obtained by culture of spinal cord pulp in vitro. FIG. 3, gr. 245: Monosterigma form obtained by culture of subcutaneous connective tissue. FIG. 4, gr. 245: Mold obtained by in vitro culture of leukocytes removed from centrifuged blood; formation of perithecia.

Pl. 33. - Gonidial forms of canine organic mold obtained by in vitro culture of fragments of lamellar connective tissue separating the gluteal muscles. (FIG. 1 to 6, gr. 245). - FIG. 1, 2: For-FIG. 4, 5: Forme Sterigmatocystis. - FIG. 3, 4: Forme Penicilium. - Fig. 5,5: Forme Acremonium alternatum.

Pl. 34. FIG. 1, 2, gr. 245**:** Mucor form of bovine tuberculosis cultures. FIG. 3, gr. 245: Mucor form of a cholera vibrio (Bologna vibrio). - FIG. 4, 5, gr. 245: Mucor form of human tuberculosis cultures.

Pl. 35. FIG. 1, 4, gr. 100**:** Mucor form of human tuberculosis cultures. FIG. 2, gr. 245: Mucor form of bovine tuberculosis cultures. FIG. 3, gr. 100: Mucor form of a cholera vibrio (Bologna vibrio).

Pl. 36. FIG. 1, gr. 1100**:** Forms of the mycelial branches of Mucor Truchisi (Lucet). FIG. 2, gr. 245: Form of the mycelial branches of a Mucor isolated by culture from the brain substance of a rabid dog.

Pl. 37. Gr. uniform 245. FIG. 1**:** Flow of basophilic protoplasm from the end of a broken filament of the Mucor form of the agent of icterohemorrhagic spirochetosis. - FIG. 2: Flow of basophilic protoplasm (myelin) from the end of a broken myelinated fiber of the rabbit sciatica. - FIG. 3, 4: Transverse septation lines of rabbit myelinated fibers; identical septation lines on mycelial branches of the Mucor form of bovine tuberculosis (FIG. 5), and of the agent of icterohemorrhagic spirochetosis (FIG. 6).

Pl. 38. FIG. 1, gr. 1100. FIG. 2 to 6, gr. 245. - FIG. 1**:** Mycelial branches of Mucor Truchisi (Lucet) showing incisures of the myelin fibers. FIG. 2, 3: Gonidiophore and mycelial branch of Mucor mucedo. - FIG. 4: Mycelial branch from an undetermined Mucor. - FIG. 5, 6: Incisures of the myelin fibers of the rabbit sciatica.

Pl. 39. Uniform gr. 245. FIG. 1: Mycelial branch of the Mucor form of human tuberculosis cultures. FIG. 2: Myelin fibers of the calf sciatica. - FIG. 3 to 6: Mycelial branches of the Mucor form of the agent of icterohemorrhagic spirochetosis.

Pl. 40. FIG. 2, gr. 610: the other figures gr. 245. Forms of the interannular constric- tions in the myelinated fibers of the sciatic of the calf (FIG. 15), and of the rabbit (FIG. 1, 3, 5, 7, 9, 11, 13, 17, 19); the same interannular constrictions in mycelial branches of Mucor: Mucor Truchisi (FIG. 20),

Mucor Regneri (FIG. 18), Mucor of the agent of icterohemorrhagic spirochetosis (FIG. 4, 6, 8, 10, 16), Mucor from human tuberculosis cultures (FIG. 14), Mucor obtained by culture of brain pulp from a rabid dog (FIG. 2, 21).

Pl. 41. FIGS. 1 to 5, gr. 830. FIGS. 6 to 11, gr. 245. FIGS. 1 to 5**:** Interannular constrictions of the mycelial branches of a form of Aspergillus repens. FIGS. 6, 7: Myelinated fibers of the rabbit sciatica. FIGS. 8 to 11: Interannular constrictions of the filaments of Mucor mucedo (FIG. 8, 9), of Mucor indeterminate (FIG. 10) and of Mucor of the agent of spirochetosis (FIG. 11).

Pl. 42. FIG. 1, 2, 4, 6, gr. 245. FIG. 3, 5, gr. 610**.** - Nuclei of the Schwann sheath, on the rabbit sciatica (FIG. 1, 3, 4) and on the mycelial branches of a Mucor obtained by culture of cerebral pulp of a rabid dog (FIG. 2, 6), and of a Mucor corymbifer of unknown origin (FIG. 5).

Pl. 43. Uniform gr. 245**:** Fructifications of myelinated fibers from the rabbit sciatica (FIG. 1, 3, 4, 5, 6), and of the mycelial branches of Mucor in vitro (FIG. 2, 7, 8). - Mucor from human tuberculosis cultures (FIG. 2), Mucor isolated from the brain of a rabid dog (FIG. 7, 8).

Pl. 44. Uniform gr. 245: Fructifications of myelinated fibers from the rabbit sciatica (FIG. 1 to 5).

Pl. 45. Uniform gr. 1100: Spiral appendages of myelinated fibers from the rabbit sciatica and their ramifications (FIG. 2, 3, 5), and of the mycelial branches of Mucor Truchisi (Lucet) (FIG. 1, 4).

Pl. 46. FIG. 1, 4, gr. 1100. FIG. 2, 3, gr. 610**:** Terminal (FIG. 1) and lateral (FIG. 3, 4) ramifications of the myelin fibers of the rabbit sciatica. Spiral appendages on the mycelial filaments of the Sporothricum form of the organic mold of humans in vitro (FIG. 2).

Pl. 47. FIG. 2, gr. 245. FIG. 1, 3, 4, gr. 610: Ramifications of the myelin fibers of the rabbit sciatica (FIG. 1, 3) and the mycelial branches of Mucor corymbifer of unknown origin (FIG. 2, 4).

Pl. 48. FIG. 1, 2, 3, gr. 245. FIG. 4, gr. 610. FIG. 1: Various elements of a culture of the Mucor form of bovine tuberculosis cultures. - FIG. 2: Myelinated fibers of the rabbit sciatica. - FIG. 3: Ramifications of the mycelial branches of Mucor corymbifer. FIG. 4: Mycelial dilations of a myelinated fiber of the calf sciatica.

Pl. 49. FIG. 1, 3, 4, gr. 245. FIG. 2, 5, gr. 610: Various aspects of the ramifications of the mycelial branches of Mucor in vitro: Mucor of the icterohemorrhagic spirochetosis (FIG. 1, 3); Mucor from the brain of a rabid dog (FIG. 4); Mucor corymbifer (FIG. 2, 5).

Pl. 50. FIG. 2, 3, gr. 100. - FIG. 1, 6, gr. 245. - FIG. 4, gr. 610. — FIG. 5, gr. 750.- FIG. 4, D, D: Lateral ramifications of the cylinder-axis of rabbit myelin fibers. - FIG. 5, 6: Lateral ramifications of the cylinder-axis of a Mucor branch in vitro (Mucor isolated from the brain of a rabid dog). - FIG. 1, 2, 3: Ramifications of an initial mycelial branch of a Mucor culture (FIG. 3) into several branches (FIG. 2) which, themselves, ramify to form a large bundle of mycelial branches (FIG. 1).

Pl. 51. Uniform gr. 245. FIG. 1, 2, 3, 5: Branching and fibrillar constitution of the axis-cylinder of the myelinated fibers of the rabbit sciatic nerve. FIG. 4: Branching or fibrillar division of the axis-cylinder of a Mucor branch isolated from the brain of a rabid dog. FIG. 5: Mycelial branches of an undetermined Mucor; longitudinal striation.

Pl. 52. FIG. 1, gr. 245. - FIG. 2, gr. 500: Branching and fibrillar structure of the axis-cylinder of the mycelial branches of Mucor in vitro (Mucor isolated from the brain of a rabid dog).

Pl. 53. FIG. 1, 3, gr. 135. FIG. 2 and 4 to 8, gr. 175: Various forms of multipolar cells (perithecia) in the grey matter of the ox spinal cord.

Pl. 54. FIG. 1, 2, gr. 1100: Constituent elements of the cat spinal cord (Imp. silver nitrate). - FIG. 3, 4: Various forms of nerve cells (perithecia) in the grey matter of the ox spinal cord.

Pl. 55. Gr. 60: A large mycelial branch of the grey matter of the ox spinal cord and its ramifications.

Plate 56-57. Gr. 91: Constituent elements of the cat's spinal cord. Cross section (Silver nitrate print).

Plate 58. FIG. 3, gr. 610. - FIG. 1, 2, 4, 5, gr. 1100: Constitution and nature of the nerve cells (perithecia) of the grey matter of the cat's spinal cord (Silver nitrate print).

Plate 59. - FIG. 5, gr. 245. FIG. 1, 2, 3, 4, gr. 1100: Same legend as Plate 58.

Plate 60. FIG. 1 to 4, gr. 245. - FIG. 1, 2: Lateral spiral appendages and terminal spiral ramifications of the mycelial filaments of the white matter of the spinal cord of the ox (dahlia stain).—Fig. 3: Fruiting bodies of the mycelial filaments of the spinal cord of the ox (dahlia stain). — Fig. 4: Cephalosporium acremonium-like mold isolated from the cerebral substance of the hemispheres of a rabid dog.

Pl. 61, 62, 63. FIG. 1 to 5, pl. 61, gr. 245. FIG. 1, 2, 3, 4, 6, pl. 62, gr. 245. FIG. 1 to 10, pl. 63, gr. 245: Perithecia shapes identical to the cells of the nerve centers in a mold isolated by in vitro culture from the root of a hair of a patient suffering from microsporia. -FIG. 5, pl. 62, gr. 1100: Perithecia of an in vitro culture of the pathogenic mold which is the agent of malaria.

Pl. 64. FIG. 1 to 5, gr. 100. FIG. 6, gr. 245: Perithecia formed in vitro by a mold obtained by culturing and transforming the pulp of the brain matter of a newborn who died at term.

Pl. 65. FIG. 1, 2: Photograph and enlargement of figure 152 from Prenant's Treatise on Histology (appendages and varicosities of the branches emanating from the cells of the cerebral cortex of the cat). FIG. 3: Purkinje cells of the cerebellum (FIG. 152, Treatise on Histology, by Prenant).

Pl. 66. Enlargement of a region of figure 430 from Ramon y Cajal's Treatise on Histology of the Nervous System (horizontal and anteroposterior section of the cerebral vesicle of a urodele).

Pl. 67. - FIG. 1, 3, gr. 91**:** Mycelial branches of the pyramidal cells of the cortex of the cerebral hemispheres of the sheep. FIG. 2, gr. 245: Fruiting bodies of mycelial filaments of the cerebral cortex of the rabbit.

Pl. 68. FIG. 1, gr. 61**:** Mycelial branches of the pyramidal cells of the cortex of the cerebral hemispheres of the sheep. - FIG. 2, 3, gr. 60: A large cell of the gray matter of the corpus striatum of the sheep and the ramified mycelial branches it emits.

Pl. 69. - FIG. 1, gr. 135: Purkinje cell of the cerebellum of the sheep -FIG. 2, 3, gr. 100: Mycelial branches and encapsulated balls of cells from the cerebellar cortex of the sheep.

Pl. 70. FIG. 1, gr. 175: Mycelial branches with their attached lateral spores, in the grey matter of the spinal cord of the ox. - FIG. 2, gr. 175: Mycelial branches in the cerebellar cortex of the sheep and the spores they form. -FIG. 3, gr. 175: Special, unknown cells and fine mycelial filaments emanating from them, in the cerebellar cortex of the sheep. -FIG. 4, gr. 60: Large mycelial branch or very long sclerotium with its mycelial branches in the cerebellar cortex of the sheep.

Pl. 71. -FIG. 1, gr. 60. -FIG. 5, gr. 175. -FIG. 2, 3, 4, gr. 245. -Fig. 1, 2, 3,: -FIG. 1, 2, 3: Encapsulated balls of the grey matter of the sheep's cerebellum. FIG. 4: Formation of spores, spiral appendages, and bunches of spiral appendages by the mycelial branches of the grey matter of the sheep's cerebellum. - FIG. 5: Large mycelial trunks and their ramifications in the grey matter of the sheep cerebellum.

Pl. 72.-FIG. 4, 5, gr. 175. FIG. 1 to 4, FIG. 6, gr. 245. FIG. 7, gr. 325. FIG. 1 to 5: Encapsulated balls of cortical gray matter from the sheep cerebellum. FIG. 6: Pear-shaped fruiting bodies of a rudimentary culture of the Penicilium form of the human organic mold. FIG. 7: Perithecium of a mold culture of the gonidial form Corymbisporium obtained by culturing a fragment of human tuberculous lung.

Pl. 73. FIG. 1, 3, 5, 6, 7, gr. 175. FIG. 4, 8, 9, gr. 245. FIG. 2, gr. 610. FIG. 1: Mass of agglomerated spores germinating in the gray matter of the ox spinal cord. -FIG. 2: Masses of agglomerated spores germinating in an in vitro culture of Mucor corymbifer. FIG. 3, 4, 5: Germinative masses of the gray matter of the sheep cerebellum. FIG. 6: Terminal spherical fructification of a mycelial branch of the gray matter of the sheep cerebellum. - FIG. 7: Ibid. Spindle formations. -FIG. 8: Round perithecia of a mold in vitro (vegetative form of Microsporum). FIG. 9: Ball- or sac-shaped fruiting bodies of a vegetative form of rat organ mold, in vitro.

Pl. 74. FIG. 1, 2, 3, gr. 245: Mold elements of the gray matter of the sheep's cerebellum. Large mycelial branches, mycelial dilations, fructifications (fruiting bodies).

Pl. 75. FIG. 1, 3, gr. 610. FIG. 2, 4, gr. 1100: End plates of the snake muscles.

Pl. 76. FIG. 1, gr. 1440. -FIG. 2, 3, gr. 1100: Composition of the sarcolemma and the motor end plates of the intercostal muscles of the rabbit.

Pl. 77. FIG. 1 to 4, gr. 1100. FIG. 5, gr. 1440: Same legend as Pl. 7.6

Pl.78.- FIG. 1, gr. 500: Dissociation of the elementary fibrils of the muscle fibers of the carp. turtle. FIG. 2, gr. 245: Idem, muscles of the FIG. 3, gr. 245: Idem, muscles of the sheep. FIG. 4, gr. 245: Torn bundle of muscle fibers of the carp showing the striation proper to the sarcolemma.

Pl. 79. FIGS. 1 to 5, gr. 1100. FIG. 6, gr. 610: Composition of the elementary fibrils of the muscle fibers of the snake.

Pl. 80. FIG. 1, gr. 245. FIGS. 2, 5, 6, 7, gr. 610. FIG. 3, 4, gr. 1100: Composition of the muscle fibers of the snake.

Pl. 81. FIGS. 1 to 4, gr. 1100**:** Composition of the muscle fibers of the snake.

Pl. 82. FIGS. 1 to 8, gr. 1100: Composition of the muscle fibers of the thigh of the guinea pig fetus

Pl. 83. FIG. 1, sq. 1440. -FIG. 2 to 9, sq. 1100. -FIG. 10, 11, gr 245**:** Constitution of the muscles of the fetal thigh of the guinea pig.

Pl. 84. FIG. 1, 4, 5, sq. 1100.-FIG. 2, 3, sq. 610. Fig. 1, 4: Constitution and formation of the muscle fibers of the fetal guinea pig. -FIG. 5: Constitution of the muscle fibers of the rat heart.

Pl. 85. FIG. 1, 3, sq. 100. -FIG. 2, sq. 60. -FIG. 4, sq. 1100: Constitution of the muscle fibers of the rat heart.

Pl. 86. FIG. 1, 2, 4, gr. 1100. FIG. 3, gr. 245: Composition of the muscle fibers of the rat's heart.

Pl. 87. FIG. 1, 3, 4, 6, gr. 610. FIG. 2, 5, gr. 1100: Composition of the muscle fibers of the rat's heart. Sarcolemma.

Pl. 88. FIG. 1, gr. 60. FIG. 2, 4, gr. 110. Constitution of the guinea pig liver. — Mycelial branches, their ramifications and their fructifications.

Pl. 89.-FIG. 1, gr. 110. FIG. 2, gr. 245: Same legend as Pl. 88.

Pl. 90.- FIG. 1 to 4, gr. 245. FIG. 1: Development of the mycelial branches and formation of hepatic cells by their ramifications.-FIG. 2, 3, 4: Groups of hepatic cells or lobules (guinea pig liver).

Pl. 91. FIG. 1, gr. 1100. FIG. 2, gr. 610. FIG. 3, 4, 5, gr. 245**:** Constitution of hepatic cells (guinea pig liver).

Pl. 92. Uniform gr. 1100: Constitution of hepatic cells (guinea pig liver).

Pl. 93. FIG. 1, 2, 3, gr. 100**:** Constitution of the sheep's spleen. Formative mycelial branches and their development.

Pl. 94. - FIG. 1 to 4, gr. 245: Constituent elements of the sheep's spleen.

Pl. 95. FIG. 1, gr. 33. FIG. 2, gr. 100. FIG. 8, gr. 245: Constitution of the guinea pig testicle. Formative mycelial branches.

Pl. 96. FIG. 1, gr. 100. FIG. 2, gr. 245: Constitution of the guinea pig testicle. Constitution of the formative mycelial branches.

Pl. 97. FIG. 1 to 5, gr. 245: Constituent elements of the guinea pig testicle.

Pl. 98. FIG. 1, 2, gr. 245. FIG. 3, 4, gr. 610: Constituent elements of the guinea pig testicle.

Pl. 99. guinea pig. FIG. 1 to 4, gr. 610: Constituent elements of the guinea pig testicle.

Pl. 100. -FIG. 1, 2, 3, 4, gr. 1100. FIG. 5, 7, gr. 610. FIG. 6, gr. 245: Constituent elements of the guinea pig testicle. Formation of spermatozoa.

Pl. 101. FIG. 1, gr. 245. FIG. 2, 3, 5, 6, 7, gr. 610. FIG. 4, 8, gr. 1100**:** Constitution of the guinea pig ovary.

Pl. 102. FIG. 1, gr. 100. FIG. 2, 3, gr. 245. FIG. 4, gr. 500: Constitution of the dog's ovary. Photograph of a section of the ovary.

Pl. 103. FIG. 1, 2, 3, 5, gr. 245. FIG. 4, gr. 610: Formation of the egg in the dog's ovary. Mode of formation of the perithecia.

Plate 104. FIGS. 1 to 6, gr. 245: Stages in the formation of the egg in the ovary of the female dog.

Plate 105. FIGS. 1 to 7, gr. 245: Stages in the formation of the egg in the ovary of the female dog (continued from Plate 104).

Plate 106. FIGS. 1 to 6, gr. 1100: Ovary of the female dog. Constitution of the ovum.

Plate 107. FIGS. 1 to 9, gr. 1440. FIGS. 1 to 7: Stages in the formation of blood corpuscles in the Alligator mississipiensis. - FIGS. 8, 9: Hemolysis of blood corpuscles by carbolic fuchsin.

Plate 108. FIGS. 1 to 5, gr. 1440 -FIG. 6, 7, gr. 1100. FIG. 1, 2, 3, 5, 6, 7: Composition of dried guinea pig blood cells colored by dahlia. -FIG. 4: Composition of intact, living guinea pig blood cells colored by dahlia.

Pl. 109. FIG. 1 to 4, gr. 735: Composition of the sheep's spleen.

Pl. 110. sheep. FIG. 1 to 7, gr. 1100: Constituent elements of the spleen of sheep.

Pl. 111. FIG. 3, 8, 11, gr. 245: The other figures, gr. 1100: Figurative elements contained in oxalated dog blood plasma.

Pl. 112. FIG. 1, 2.5, gr. 245. FIG. 3, gr. 100.- FIG. 4, gr. 1440.-FIG. 6, 7, gr. 610: Composition of the blades of the torpedo's electrical organ.

Pl. 113. FIG. 1 to 7, gr. 245: In vitro transformation of the organic mold of the torpedo's electric organ into Aspergillus-like mold.

Pl. 114. FIG. 1, 2, 3, 4, 9, gr. 1100. FIG. 5, 6, 7, gr. 245. - FIG. 8, gr. 735: Constitution of the diastase of germinated barley. Vegetation of the ferment and stages of its evolution in broth.

Pl. 115. FIG. 1 to 4, gr. 1100. FIG. 5, 7, 8, 9, gr. 245. - FIG. 6, gr. 175: Constitution of pork pepsin. Vegetation of the ferment in broth and stages of its evolution.

Pl. 116. FIG. 4, gr. 1440, all other figures gr. 1100. - FIG. 1: Constitution of tetanus toxin. Zymospores. - FIG. 2: Evolution of tetanus toxin and its transformation in bacillary culture.- FIG. 3: Constitution of diphtheria toxin. Zymospores. FIG. 4 to 9: Evolution of the elements of diphtheria toxin and stages of their transformation in bacillary culture. - FIG. 10, 11: Origin of the zymospores of the toxin; elements of a diphtheria culture in a pure glucose solution.

Plate 117. FIG. 1, 2, gr. 245. FIG. 3, gr. 610. FIG. 4, gr. 1100: Composition of the flakes that form in fibrinogen solutions of horse blood.

Plate 118. FIG. 1 to 7, gr. 610: Same legend as plate 117 (fibrinogen solution in the process of coagulation).

Plate 119. FIG. 1, 2, gr. 245. Plate 117. FIG. 3, 4, gr. 100: Same legend as Plate 117.

Plate 120. -FIG. 1, gr. 245. -FIG. 2, 3, 4, gr. 610. -FIG. 5 to 9, gr 1100.- FIGS. 1 to 4: Colonies of spores growing in a solution of purified fibrinogen, spontaneously coagulated. FIGS. 5 to 9:

Constituent elements of a guinea pig blood clot crushed between two glass slides and stained with dahlia.

Pl. 121. FIG. 1, 2, 3, gr. 1440: Disorganization and evolution of guinea pig blood cells in the presence of rabbit complement and the precipitate of globulins from a rabbit anti-guinea pig red blood cell serum. -FIG. 4, gr. 1100: Elements of the guinea pig blood clot crushed between two glass slides and stained with dahlia. -FIG. 5, 6, gr. 1100: Bacterial elements from the broth culture of the elements contained in the leukocyte layer of oxalated and centrifuged horse blood.

Pl. 122. -Photographs demonstrating the existence of agglutinating hyaline matter (bacterial fibrin) deposited on the bacterial elements and determining their adhesion in clusters.

Agglutination, FIG. 1, 2, gr. 1100: Elements of a broth culture of the Penicilium form of rennet reduced to the bacterial state; the hyaline matter constitutes the faded spots. FIG. 4: Diagram of the clusters showing the position of the bacterial elements in the clusters; the voids separating them are occupied by the agglutinating hyaline matter. -FIG. 3, gr. 1100: Bacterial culture of lettuce organic mold showing the presence of agglutinating hyaline matter in the bacterial clusters.

Precipitation, FIG. 5, gr. 1100: Formation of a flake (mycelial colony) in horse serum supplemented with precipitating dog serum. FIG. 6: *Idem:* flake consisting of a cluster of germinating spores, whose mycelial filaments form zymospores.

Pl. 123. FIG. 1, 2, 3, gr. 245. FIG. 1: Thin cross-section of the leek leaf (col. dahlia). -FIG. 2, 3: Thick sections of the leek leaf (impregnation with silver nitrate).

Pl. 124.- FIG. 1, 2, gr. 245: Cross-sections of the leek leaf.

Pl. 125. FIG. 1 to 6, gr. 245. FIG. 2, 3, 4, gr. 91. - FIG. 5, gr. 1100. FIG. 1: Mycelial branch of the parenchyma dissociated from the onion bud scale. - FIG. 2 to 6: Elements of the dissociated parenchyma of the leek leaf.

Pl. 126. -FIG. 1, gr. 1100. -FIG. 2, gr. 610. -FIG. 3, gr. 245: Constituent elements of the leek leaf.

Pl. 127. -FIG. 1, gr. 1100. -FIG. 2, 3, gr. 610: Elements of the leek leaf. Cross-section of the leaf impregnated with silver nitrate.

Pl. 128. FIG. 1 to 4, gr. 245. -FIG. 5 to 8, gr. 610: Elements of the leek leaf.

Pl. 129. FIG. 1, 2, gr. 610. FIG. 3, gr. 1100: Constituent elements of the leek leaf. -FIG. 1, 2: Cross-section of the leaf impregnated with silver nitrate.

Pl. 130. FIG. 1, gr. 245. FIG. 2, gr. 91. FIG. 1: Cross-section of the leek leaf impregnated with silver nitrate. FIG. 2: Cross-section of the onion bulb scale (colored by the dahlia).

Pl. 131. FIG. 1, 2, gr. 245. FIG. 3, 4, gr. 91. FIG. 1: Cross-section of the onion bulb scale impregnated with silver nitrate. FIG. 2: Longitudinal section of the onion bulb scale impregnated with silver nitrate. -FIG. 3, 4: Torn edges of a cross-section of the onion bulb scale colored by the dahlia.

Pl. 132. FIG. 1, 2, 3, gr. 245**:** Photographs of a transverse section of the onion bulb scale, in the regions of the libero-ligneous bundles (Impregnated with silver nitrate).

Pl. 133. FIG. 1, 2, 3: Constitution of the carrot root parenchyma.

Pl. 134. 9, 10, 12, gr. FIG. 1, 3, 4, gr. 610. - FIG. 2, 6, 7, 11, gr. 245. - FIG. 5, 8, 1100: Constituent elements of the carrot parenchyma.

Pl. 135. FIG. 1, 2, 3, 4, 7, gr. 245. FIG. 5, gr. 100. - FIG. 6, gr. 610: Constituent elements of the parenchyma of an apple.

Pl. 136. FIG. 1, 3, gr. 245. - FIG. 2, 4, 5, gr. 490: Constituent elements of the leaf of Althea officinalis.

Pl. 137. FIG. 1, 2, 3, gr. 245: Constitution of the parenchyma of the leaf of Althea officinalis. - FIG. 4: Stromatinia cydonia (Fig. 1, 2, page 259, Delacroix and Maublanc, Parasitic Diseases of Cultivated Plants).

Pl. 138. Roots of FIGS. 1 to 6, gr. 245: Composition of the root and root hairs of germinated wheat.

Pl. 139. FIGS. 1 to 8, gr. 245: Composition of the parenchyma of the stem of hymenomycete fungi. Collybia velutipes.

Pl. 140. FIGS. 1 to 8, gr. 245: Evolutionary forms of the fruiting bodies of a mold from the Aspergillus type to the Penicilium aspergilloide type.

Pl. 141. -Main stable gonidial forms of molds. -FIG. 1: Forme Penicilium. -FIG. 2: Forme Citromyces. -FIG. 3: Forme Aspergillus. -FIG. 4: Sterigmatocystis form. FIG. 5: Forme Mucor corymbifer. -FIG. 6: Forme Sporothricum. -FIG. 7: Forme Verticilium. -FIG. 8: Forme Fusarium. -FIG. 9: Fusophragmium form. -FIG. 10: Alternaria form. -FIG. 11: Forme Fusophragmium of Achorion Gypseum. -FIG.12: Monosterigmata form. FIG. 13: Forme evolved Monosterigmata. -FIG. 14: Forme Passalora. -FIG. 15: Form Peronospora or Botrytis cinerea (Bremia lactucae). FIG. 16: Cladosporium or Monilia form. -FIG. 17: Form Acremonium alternatum. -FIG. 18: Spicaria form.

Pl. 142. FIG. 1 to 4, gr. 175. FIG. 1, 2, 3: Constitution of starch grains in potatoes. FIG. 4: Formation of starch grains by the mycelial filaments of a Fusarium solani culture.

Pl. 143. FIG. 1, 2, 3, gr. 245: Fusarium solani. -FIG. 4, gr. 175: Formation of a perithecia (starch grain) by mycelial filaments of Fusarium solani. FIG. 5, gr. 245: Constitution of starch grains in the potato parenchyma.

Pl. 144. FIG. 1, 2, gr. 245: Mode of formation of starch grains (perithecia) in the potato parenchyma.

Pl.145. -FIG. 2, 9, 10, gr. 610. FIG. 1, 3, 4, 5, 6, 7, 8, 11, 12, gr. 1100: Spontaneous transformation of the organized mold of the parenchyma of the potato tuber into bacterial culture. -FIG. 1, 2: Bacterial elements of the spontaneous wet rot of tubers in the ground. -FIG. 3, 4, 5: Bacterial elements of the rot of the frozen potato. -FIG. 6, 7: Bacterial elements of the rot of the potato heated for one or two hours at 550, -FIG. 8: Bacterial elements of the broth culture of the pulp of the healthy fruit of the potato. -FIG. 9 to 12: Culture of Fusarium solani in liquefaction and passing to the bacterial state.

Pl. 146. FIG. 1 to 10, gr. 245: Gonidial forms of potato organic mold developed by transformation of the healthy tuber parenchyma. FIG. 1 to 3: Phytophtora infestans form. -FIG. 4, 5: Verticilium

special form. FIG. 6: Acremonium alternatum and Cephalosporium acremonium form. -FIG. 7. Spicaria form. -FIG. 8, 9, 10: Fusophragmium form.

Pl. 147. -FIGS. 1 to 10, gr. 245. -FIGS. 1 to 6: Mycelium and gonidia of one of the forms of Phytophtora infestans developing on brown rot of the potato. -FIGS. 7 to 10: Fusarium solani evolving into another gonidial form (Ovularia).

Pl. 148. -FIGS. 1 to 7, -FIG. 9, gr. 245. -FIG. 10, gr. 610. -FIG. 1 to 5: Evolution of the form Fusarium solani into the form Penicilium solani. FIGS. 6, 7, 8: Form Spicaria verticillata solani. FIG. 9: Form Acremonium alternatum solani.

Pl. 149. -FIGS. 1 to 6, gr. 245. -FIGS. 7, 8, gr. 610.- FIGS. 1 to 3: Form of Phytophtora infestans changing to the Mucor solani type. FIGS. 4, 5: Mucor solani. -FIGS. 6, 7, 8: Phytophtora infestans obtained by transformation of normal organic potato mold (passage through the guinea pig).

Pl. 150. -FIGS. 1, 3, 4, 6, gr. 245. -FIGS. 2, 5, gr. 100: Identity of Plasmopara viticola (FIG. 3), and Botrytis cinerea of grapes (FIG. 1, 2, 4), Neighboring fruiting bodies, in the same microscopic field, of a culture of Botrytis cinerea. Transformation of Botrytis into Penicilium (Fig. 6, 7).

Pl. 151. FIGS. 1 to 10, gr. 245**:** Fructifications of Bremia lactucae and its transition to other gonidian forms.

Pl. 152. FIGS. 1, 2, 6, gr. 245. FIGS. 3, 4, 5, gr. 135: Teliospores of Puccinia Malvacearum and their origin.

Pl. 153. FIGS. 1 to 8, gr. 245: Constituent elements of the parenchyma of the normal, healthy leaf of Althea officinalis. Terminal ramifications of the mycelial branches of the libero-ligneous bundles, in the parenchyma itself, in the form of gonidiophores of Botrytis cinerea.

Pl. 154. - FIG. 1 to 6, gr. 245: Sporothricum and Fusophragmium forms obtained by in vitro culture on agar of the teliospores of Puccinia Malvacearum.

Pl. 155. -FIG. 1, 3, 6 to 10, gr. 245. -FIG. 2, 4, 5, gr. 610. -FIG. 1 to 6: Gonidian forms of organic mold of wheat,-FIG. 1: Complex form Monilia, Acremonium and Sporothricum. -FIG. 2: Fusarium tritici. -FIG. 3, 4: Sclerospora form (Peronospora). - FIG. 5: Mycelial filament of the Sclerospora form bearing a Peronospora gonidia and a bilocular Fusarium gonidia. -FIG. 6: Mycelial filaments of the Sclerospora form forming stylospores and sporidia identical to those that develop by germination of Tilletia tritici spores. -FIG. 7: Alternaria form. FIG. 8, 9: Normal parenchyma of the young wheat leaf. Normal cells of the uredospore and teliospore forms of Puccinia glumarum.

Pl. 156. FIG. 1, gr. 245. FIG. 2 to 10, gr. 610. FIG. 1: Constituent elements of a carious wheat grain (spores of Tilletia tritici). -FIGS. 2 to 10: Gonidian forms obtained by culture of the spores of Tilletia tritici. -FIGS. 2 to 7: Fusophragmium form, with two-celled gonidia (teleutospores), stylospores, etc. -FIGS. 8 to 10: Penicilium form.

Pl. 157.-FIGS. 1, 3, gr. 245. -FIGS. 2, 4, gr. 610**:** Constitutive elements of the parenchyma of the young Mars leaf.

Pl. 158. FIGS. 1 to 10, gr. 245: Puccinia Maydis. -FIGS. 1, 2: teliospores of the sori. - FIGS. 3 to 5: Elements of a uredo. - FIGS. 6 to 9: Sporothricum form contained in the uredos. FIG. 10: Spores of smut growing on the leaves.

Pl. 159. -FIG. 1, 2, 6, 7, 9, 10, 11, gr. 610. -FIGS. 3, 4, 5, 8, 13, 14, 15, gr. 245.-FIG. 12, gr. 33. - FIGS. 1 to 7: Gonidian forms obtained by culturing the spores of a smut gall of corn. -FIG. 12: Fusophragmium form (?). - FIG. 4: Sporothricum form.-FIG. 3, 5: Cephalosporium acremonium form. — FIG. 6, 7: Penicilium form. - FIG. 8 to 15: Gonidian forms of organic corn mold obtained by culture of healthy stem tissue. - FIG. 1: Complicated form Monilia, Acremonium, Sporothricum. - FIG. 9 to 11: Penicilium form. -FIG. 12, 13: Mucor corymbifer form. -FIG. 14, 15: Fusarium form.

Pl. 160.-FIGS. 1 to 5, gr. 245.-FIG. 6, gr. 100.-FIGS. 7 to 10, gr. 610: Oat smut. -FIG. 1: Monosterigma gonidial form of oat smut growing on grains. -FIGS. 2, 3: The same Monosterigma form obtained by passage of smut spores through the guinea pig's body.- FIGS. 4, 5: Smut spores growing on oat ears.-FIGS. 6 to 9: Aspergillus fumigatus forms(FIG. 6) and Penicilium, obtained by passage of oat smut spores through the guinea pig's body.

Pl. 161.-FIG. 1, gr. 175.-FIG. 2 to 8, gr. 245.-FIG. 9, gr. 100.-FIG. 10 to 13, gr. 610. -FIG. 1: Mycelium of the gonidial form Monosterigmata vegetating along the hairs of the glumes. -1, FIG. 2: Vegetation of the same form on the glumes. -FIG. 5: Cladosporium forms or Monilia and Mucor form (FIG. 8, 9) obtained by direct culture of anthrax spores on agar. -FIG. 3, 4: Monosterigmata form. - FIG. 6, 7: Rudimentary Penicilium form. -FIG. 10 to 13: Perfect Penicilium form obtained by culturing the organs of the guinea pig inoculated with anthrax spores.

Pl. 162. FIG. 1, 2, 4, 5, 6, gr. 245. FIG. 3, gr. 610: Penicillium form obtained by direct transformation of a human tuberculosis culture in vitro.

Pl. 163. FIG. 1, 2, gr. 100. FIG. 2, 4, gr. 245. FIG. 5, gr. 610: Mucor racemosus (FIG. 1, 2, 3), and Sterigmatocystis (FIG. 4, 5) forms obtained by direct transformation of a human tuberculosis culture in vitro (Source: Pasteur Institute).

Pl. 164. FIG. 1 to 16, gr. 610: Different types of the Penicillium form obtained by direct transformation of a human tuberculosis culture (Source: Pasteur Institute).

Pl. 165.-FIG. 1 to 16, gr. 610: Same legend as Pl. 164 (continued).

Pl. 166. FIG. 1 to 16, gr. 610: Different types of the Penicillium form obtained by transformation of a culture of human tuberculosis (Culture Ay. Origine, Küss).

Pl. 167. FIG. 1 to 15, gr. 610: Same legend as Pl. 166 (Lacr. culture, Origin: Kuss).

Pl. 168. -FIG. 1, gr. 245. FIG. 2, 3, gr. 610: Different types of the Penicilium form obtained by direct transformation of a bovine tuberculosis culture (Origin: Vallée).

Pl. 169. - FIG. 1, 2, 3, gr. 245. FIG. 4, 5, 6, gr. 100: Aspergillus (FIG. 1), Mono-sterigmata (FIG. 2) and Mucor (FIG. 3 to 6) forms obtained by indirect transformation of a bovine tuberculosis culture (Origin: Vallée).

Pl. 170.- FIGS. 1 to 22, gr. 610: Different types of the Penicilium form obtained by direct transformation of a bovine tuberculosis culture (Origin: Vallée).

Pl. 171.-FIGS. 1 to 14, gr. 610: Same legend as Pl. 170 (continued) (Origin: Vallée).

Pl. 172. FIG. 1, 2, gr. 610. FIG. 3, 4, 5, gr. 245. - FIG. 1, **2:** Penicilium form obtained by direct transformation of a culture of avian tuberculosis (Origin: Pasteur Institute). - FIG. 3, 4: Aspergillus form. - FIG. 5: Monosterigmata form, obtained by indirect transformation of the same culture (passage through the guinea pig).

Pl. 173. - FIG. 3 to 7, gr. 610. - FIG. 1, 2, FIG. 8 to 16, gr. 245.: Different gonidial forms obtained by culture of lung tissue and the caseous material of human tubercles. FIG. 1: Penicilium. -FIG. 2 to 6: Citromyces. FIG. 7, 8: Acaulium. -FIG. 9, 10: Aspergilus minimus. - FIG. 11, 12: Sterigmatocystis. - FIG. 13, 14: Mono-sterigmatis. - FIG.15,16: Cephalo- sporium acremonium or Acremonium alternatum.

Pl. 174. FIG. 1 to 16, gr. 610: Different types of the Penicillium form of the human organic mold (dead newborn). FIG. 8 and 11 to 16: Types obtained from the pulp of the liver; the other types from the pulp of lung tissue.

Plate 175. Continuation of Plate 174. FIGS. 1, 5, 8, 18, 15, 19, 20: Types obtained from the liver pulp; the other types from the lung pulp (uniform Gr. 610).

Plate 176.-FIGS. 1 to 7, Gr. 610.-FIGS. 8 to 12, Gr. 245**:** Different gonidial forms of the organic mold of man (newborn dead at term). Citromyces form obtained from the cerebral pulp of a newborn (FIG. 1, 2), and from the pulp of the lung tissue (FIG. 3 to 7) of another newborn. FIG. 8: Sterigma-tocystis form obtained from the lung pulp. FIG. 9, 11, 12: Sporothricum form obtained from the cerebral pulp of a newborn (FIG. 9), the lung tissue of a second newborn (FIG. 11), and the cerebral pulp of a third (FIG. 12). FIG. 10: Little-developed Aspergillus form obtained from the lung pulp of a newborn.

Pl. 177. FIG. 1 to 16, gr. 610: Different types of the Penicilium form of the organic mold of beef. FIG. 1, 2, 3, 5, 15: Types obtained from the liver pulp, the other types from the lung pulp.

Pl. 178. FIGS. 1 to 12, gr. 610. FIGS. 13, 14, 15, 16, gr. 100. - FIGS. 1 to 2**:** Different types of the Penicilium form of the organic mold of beef. FIGS. 13, 14: Mucor form. FIGS. 15, 16: Rhizopus form of the organic mold of beef.

Pl. 179. FIGS. 1 to 6, gr. 1100: Constituent elements of the veil (germinal masses or leukocytes) of two broth cultures (FIG. 1 to 3, culture no. 1; FIGS. 4 to 6, culture no. 2) obtained by spontaneous transformation of the lung tissue of two newborns cultivated on agar and transferred to broth (20-day culture).

Pl. 180. FIG. 1 to 8, gr. 1100: Constituent elements of the veil of a broth culture obtained by subcul-turing the culture of the newborn's lung tissue on agar. Elements of the veil after ten hours (FIG. 1, 2, 3) and thirteen hours (FIG. 4, 5, 6). - FIG. 7: Bacterial culture of breast cancer, containing the bacillary form growing in the cancerous tissue itself. Formation of a germinative mass and a large mycelial branch by germination. - FIG. 8: In vitro transfor- mation into germinative masses of the caseous matter extracted from a tubercle of the human lung. Elements of a three-day broth culture, obtained by subculturing the veil whose elements are represented in Pl. 181.

Pl. 181.-FIG. 1 to 5, gr. 1100: Constituent elements of the veil of a forty-five-day broth culture obtained by transformation of the caseous matter of a tubercle of the human lung.

Pl. 181. FIG. 1 to 5, gr. 1100: Constituent elements of the veil of a forty-five-day broth culture obtained by transformation of the caseous matter of a tubercle of the human lung.

Pl. 182. FIG. 1, gr. 175. FIG. 2, gr. 245. FIG. 3, 4, gr. 1100. -FIG. 1: Mycelial vegetation in a caseous tubercle. -FIG. 2: Elements of the lung tissue bordering a cavern. - FIG. 3, 4: Transformation of a normal veil of a human tuberculosis culture, by culture at 200, in non-glycerinated broth. Elements of the newly formed veil and transformation of the culture into the Monosterigmata gonidial form.

Pl. 183. FIG. 1, 2, 6, 7, 8, 9, gr. 1100. - FIG. 3, 4, 5, gr. 610. -FIG. 1 to 5**:** Composition of the caseous matter of the tubercles of the human lung; germinative masses and mycelial filaments they emit. FIG. 9: Germination of the spores of the caseous matter and formation of zymospores by the mycelial filaments they emit. -FIG. 6, 7, 8: Germinative masses and their mycelial branches in a culture of tuberculosis on potato.

Pl. 184. FIG. 1, gr. 650.- FIG. 2, gr. 245. FIG. 3, gr. 610: Constituent elements of cancerous tissue (uterine cancer no. 1) (FIG. 1: Drawing. - FIG. 2, 3: Photographs).

Pl. 185. FIG. 1, gr. 650 to 700 (drawing). - FIG. 2, 3, gr. 610 (Photograph): Same caption as Pl. 184 (continued).

Pl. 186. FIG. 1, gr. 245. FIG. 2, 3, gr. 610: Constituent elements of cancerous tissue (uterine cancer no. 1).

Pl. 187.-FIG. 1, 2, 3, gr. 245. FIG. 4, 5, 6, gr. 610. FIG. 1: Constituent elements of cancerous tissue (uterine cancer no. 1). - FIG. 2 to 6: Sterigmatocystis form mold obtained by in vitro culture of cancerous tissue (uterine cancer no. 1).

Pl. 188. FIG. 1, gr. 650 (except sclerotium no. 1, gr. 235). - FIG. 1: Constituent elements of cancerous tissue (uterine cancer no. 2). - FIG. 2 to 6, gr. 610: Penicilium form obtained by culturing cancerous tissue.

Pl. 189. FIG. 1 to 9, gr. 610: Molds obtained by culturing cancerous tissue (uterine cancer no. 3). FIG. 1 to 3: Sporothricum form and its evolution to the Penicilium form. FIG. 4 to 9: Penicilium form.

Pl. 190. FIG. 1, gr. 130. FIG. 2, gr. 650: Constituent elements of cancerous tissue (breast cancer no. 1).

Pl. 191. FIG. 1, gr. 690. FIG. 2, gr. 245. -FIG. 1: Constituent elements of cancerous tissue (breast cancer, no. 1). - FIG. 2: Sporothricum form mold obtained by in vitro culture of cancerous tissue.

Plate 192. Continuation of plate no. 191. - FIG. 1, 2, 4, gr. 245. - FIG. 3, gr. 610. - FIG. 1, 2, 3: Penicilium form FIG. 4: Monosterigmate form (grey mold) of vegetating syphilis in breast cancer.

Plate 193. FIG. 1, gr. 655. FIG. 2, gr. 53. -FIG. 3, 4, gr. 245.-FIG. 3, 4: Constituent elements of cancerous tissue (breast cancer no. 2). - FIG. 4, 5: Mold of the Acremonium alternatum form obtained by in vitro culture of cancerous tissue.

Plate 194. -FIG. 1 (drawing), approximate gr. 700. -FIG. 2 to 6, gr. 610. -FIG. 1: Constituent elements of cancerous tissue (breast cancer no. 3). - FIG. 2 to 6: Penicilium mold obtained by in vitro culture of cancerous tissue.

Plate 195. -Drawing, approximate gr. 700: Constituent elements of cancerous tissue (breast cancer no. 4).

Plate 196. Continuation of plate 195 (breast cancer no. 4). - FIG. 1, gr. 235: Elements of cancerous tissue. - FIG. 2 to 7, gr. 610: Penicilium mold obtained by in vitro culture of cancerous tissue.

Pl. 197. FIG. 1 to 4, gr. 245: Mold presenting the double type Sterigmatocystis and Aspergillus, obtained by culture of cancerous tissue (breast cancer no. 5).

Pl. 198. FIG. 1, gr. 245. FIG. 2, 3, gr. 610. - FIG. 4 to 11, gr. 610.- FIG. 1 to 3: Penicilium form obtained by in vitro culture of breast cancer No. 6. FIG. 4 to 11: Penicilium form obtained by in vitro culture of breast cancer tissue No. 7.

Pl. 199. FIG. 1, 2, 3, 4, 7, gr. 610. FIG. 5, 6, gr. 245. FIG. 1, 2: Penicilium form obtained by in vitro culture of breast cancer tissue No. 8. FIG. 3, 4, 5: Penicilium form obtained by in vitro culture of breast cancer tissue No. 9. -FIG. 6: Sporothricum form obtained by in vitro culture of cancerous tissue from a recurrent tumor of the larynx region. -FIG. 7: Monosterigmata form obtained by in vitro culture of breast cancer tissue No. 10.

Pl. 200. FIG. 1 to 7, gr. 1100: Transformation of normal elements of the tissue of the newborn's spleen into elements characteristic of cancerous tissue. Constituent elements of the veil of a twenty-five-day broth culture, obtained by transfer of a culture of the newborn's spleen onto agar.

Pl. 201.- Pneumonia. - FIGS. 1 to 5, gr. 245: Elements of human organic mold growing in the sputum of a pneumonia patient.

Pl. 202. FIGS. 1, 2, 4, gr. 245. FIGS. 3, 5, gr. 610: Penicillium form obtained by culture on agar of pneumonia patient's sputum.

Pl. 203.- Bacterium coli (FIG. 1, gr. 630. FIG. 2, 7, 8, gr. 610. -FIG. 3: gr. 1130. FIG. 4, 5, 6, gr. 245). -FIG. 1, 2: Citromyces form of bacterial cultures of Bacterium coli. FIG. 3: Bacterial elements (Bacterium coli) formed in the culture of Citromyces. FIG. 4 to 8: Penicilium form of bacterial cultures of Bacterium coli.

Pl. 204. - Actinomycosis (Nocardia bovis) (FIG. 1, gr. 830. - FIG. 2, gr. 245. FIG. 3, gr. 610). Mycelium and its fruiting bodies constituting the veil of a broth culture. FIG. 2, 3: Penicilium form in agar culture.

Pl. 205. Septic vibrio (FIG. 1, gr. 135. FIG. 2, gr. 830. FIG. 3, 4, gr. 245. FIG. 5, gr. 610). - FIG. 1, 2: Corymbisporium form of the septic vibrio. - FIG. 3, 4, 5: Penicilium form.

Pl. 206. - Tetanus, chickenpox, typhoid fever and corn (FIG. 1 to 5, gr. 610.FIG. 6 to 13, gr. 1100. FIG. 1 to 5: Peronospora form of the tetanus mold. - FIG. 6: Bacterial form obtained by transformation of the tetanus toxin upon contact with air. - FIG. 7 to 10: Elements of bacterial cultures of the chickenpox pathogen (7, ten-day broth culture; 8, three-day culture; 9, 10, ten-day culture). - FIG.

11, 12: Twenty-four-hour bacterial culture obtained by transformation of the leaf parenchyma of sown corn FIG. 13: Transformation form of the typhus bacillus in broth. cultivated in broth (elements of the veil, tenth day).

Pl. 207. Tetanus (FIG. 1, 2, 6, 9, gr. 610. FIG. 3, 4, 5, 7, 8, 9, gr. 245). - FIG. 1, 2: Corymbisporium form of the tetanus mold. - FIG. 3, 4: Sterigmatocystis form. FIG. 5 to 9: Penicilium form.

Pl. 208. Spontaneous tetanus in humans (FIG. 1, gr. 1100. FIGS. 2, 3, 9, 10, gr. 610. FIGS. 4 to 8, gr. 245). - FIG. 1: Elements of the tetanus mold growing in the blood. FIG. 2: Mold obtained by transformation of a broth culture. -FIG. 3 to 6: Evolution of the veil of a broth culture towards the Aspergillus form. -FIG. 7 to 10: Penicilium form.

Pl. 209. Spontaneous tetanus (continuation of plate 208). FIG. 1, 2, 6, 7, gr. 245. -FIG. 3, gr. 610. FIG. 4, 5, gr. 33). Mold obtained by indirect transformation of the gonidial forms of tetanus. -FIG. 1, 2, 3: Penicilium form. FIG. 4, 5: Mucor form. FIG. 6, 7: Penicilium form.

Pl. 210. - Leprosy (FIG. 1, gr. 245.-FIG. 2, 3, 4, gr. 610). - FIG. 1:. Mycelial vegetation constituting a leproma. - FIG. 2, 3, 4: Penicilium form obtained by in vitro culture of leproma tissue.

Pl. 211. Glanders (FIG. 1, 3 to 7, gr. 610: Penicilium form of B. mallei. - FIG. 2, gr. 245: Aspergillus form.

Pl. 212.- Diphtheria (Fig. 1, 2, 3, 6, 7, gr. 245. FIG. 4, 8, gr. 610. - FIG. 5, gr. 535). - FIG. 1 to 5: Citromyces form of a diphtheria bacillus culture. FIG. 6, 7, 8: Citromyces form obtained by culture of false diphtheria membrane (no. 6) on agar.

Pl. 213. Diphtheria. - FIG. 1, 5, gr. 610. FIG. 2, 3, 4, gr. 245: Penicilium (FIG. 1) and Monosterigmata (FIG. 2) forms obtained by culture of false membrane (no. 8) on agar. - FIG. 3, 4, 5: Penicilium form obtained by culture of false membrane (no. 7) on agar.

Pl. 214. Diphtheria. FIGS. 2 to 11, gr. 610. FIG. 1, gr. 245. -FIG. 1, 2, 4, 6, 7, 8, FIG. 11, lower half: Penicilium form obtained by culture of false membrane (no. 3) on agar. -FIG. 11, upper half: Penicilium form from a broth culture of diphtheria.

Pl. 215. -Diphtheria. - FIGS. 1 to 10, gr. 610. -FIG. 11, 12, gr. 245. Gonidial forms obtained by indirect transformation of the diphtheria toxin. FIG. 1 to 10: Penicilium form. -FIG. 11, 12: Monosterigmata form.

Pl. 216. Penicilium form of organic wheat mold. - FIGS. 1 to 8, gr. 610. - FIGS. 9, 10, gr. 245. - Penicilium form obtained by transformation of spores of decayed wheat grain (FIG. 1 to 7), or culture of pounded wheat leaves (FIG. 8, 9, 10).

Pl. 217. Penicilium form of the organic mold of wheat (FIG. 1 to 17, gr. 610), obtained by indirect transformation (passage through a guinea pig) of the spores of a decayed wheat grain (FIG. 1, 3, 4, 5, 6, 10, 11, 14), or of the pulp of the parenchyma of young wheat leaves (FIG. 2, 7, 8, 9, 12, 13, 15, 16, 17).

Pl. 218. Penicilium form of the organic mold of barley (FIG. 1 to 13, gr. 610) obtained directly by culturing the pulp of young, pounded barley leaves (FIG. 1, 3, 4, 5, 6, 8) or indirectly by transforming the spores of Ustilago hordei (passage through the guinea pig, FIG. 2, 7, 9 to 13).

Pl. 219. Penicilium form of the organic mold of rye (FIG. 1 to 17, gr. 610), obtained directly by culturing crushed rye grains.

Pl. 220.-FIG. 1 to 11, gr. 1100. — FIG. 1 to 6: Elements of bacterial cultures of organic wheat mold obtained by culturing the spores of Tilletia tritici (FIG. 1, 3, 5, 6) or the pulp of young wheat leaves (FIG. 2, 4). FIG. 7 to 11: Elements of a culture of diphtheria bacillus in broth.

Pl. 221. FIG. 1 to 12, gr. 1100.- FIG. 1 to 4: Elements of bacterial cultures of organic mold of rye (one, three, four, seventeen days) (culture of crushed rye grains). - FIG. 5 to 10: Elements of bacterial cultures of organic mold of barley (culture of crushed leaf pulp) (FIG. 9, 10, twenty-four hours, FIG. 5, 6, 7, two days. FIG. 8, ten days). - FIG. 11, 12: Bacterial elements of a diastase broth culture of germinated barley (one day and four days).

Pl. 222. Paratyphus B. FIG. 1 to 17, gr. 610: Penicilium form of cultures of paratyphus bacillus B, obtained by passage through the guinea pig.

Pl. 223. Typhoid fever. FIG. 1, gr. 1130. FIG. 2, gr. 1100. -FIG. 3, gr. 1160.- FIG. 4, gr. 245. FIG. 5, 6, gr. 610.- FIG. 1: Eberth's bacilli formed in Citromyces cultures. FIG. 2, 3: Developmental form of Eberth's bacillus on agar; germinative masses, mycelial filaments emanating from them (cilia), and zymospores they form. -FIG. 4, 5, 6: Citromyces form of Eberth's bacillus cultures.

Pl. 224. - Typhoid fever. FIGS. 1 to 15, gr. 610: Penicilium form of Eberth bacillus cultures (passage through guinea pig. Eberth verified).

Pl. 225. -Typhoid fever. FIG. 1 to 12, gr. 610: Penicilium form of Eberth bacillus cultures (passage through guinea pig. Eberth Lard.).

Pl. 226. FIG. 1 to 16, gr. 610: Penicilium form of organic corn mold, obtained directly by pulp culture of the stalk parenchyma.

Pl. 227. Exanthematic Typhus. FIG. 1 to 11, gr. 610. FIGS. 12, 13, gr. 245. - FIG. 1 to 11: Penicilium form of the pathogenic agent of Exanthematic typhus. - FIG. 12, 13, Sterigmatocystis forms.

Pl. 228. - Exanthematic typhus (continued). - FIG. 1, 2, 3, 4, 7, gr. 100. - FIG. 5, 6, gr. 610. FIG. 8, 9, 12, 13, gr. 1100.- FIG. 1 to 7: Mucor form of the pathogen. Transformation into Penicilium on the filaments of Mucor (FIG. 5, 6, 7). Transformation of Mucor into Monosterigmata form (FIG. 10, 11). - FIG. 8, 9, 12, 13: Bacterial culture of the pathogen.

Pl. 229. -FIG. 1 to 7, gr. 610. FIG. 8, 9, 10, gr. 1100**:** Penicilium form of the organic mold of oats (FIG. 1 to 7), obtained indirectly (inoculation of Ustilago aveni spores into the guinea pig). Elements of the bacterial culture of the organic mold of oats: FIG. 8: Culture of ground grains in broth; FIG. 9, 10: Culture of pulp of young oat leaves).

Pl. 230. Epidemic dysentery. - FIG. 1 to 15, gr. 610: Penicilium form of cultures of the dysentery bacillus of the Flexner type (FIG. 1 to 8) and of the Shiga type (FIG. 9 to 15).

Pl. 231. FIG. 1 to 15, gr. 610: Penicilium form of the organic mold of Brassica napus, obtained by direct culture of turnip pulp.

Pl. 232. FIG. 1 to 11, gr. 1100. FIG. 1 to 6**:** Elements of bacterial broth cultures of chicory (Cichorium intybus) one day old (FIG. 1, 2, 3), two days old (FIG. 4), four days old (FIG. 5), ten days old (FIG. 6). - Elements of a bacterial culture of radish (Raphanus sativus, var. radicula) two days old (FIG. 7, 8). - Elements of a bacterial culture of turnip (Brassica napus) two days old (FIG. 10) and four days old (FIG. 9). - Elements of a bacterial culture of cabbage (Brassica oleracea, var. capitata) two days old.

Pl. 233. FIG. 1 to 13, gr. 1100: Elements of bacterial cultures of the organic mold of red beet (Beta vulgaris) inoculated with: beet pulp (FIG. 1, two days. - FIG. 3, six days. FIG. 4, 5, 6, ten days); beet juice (FIG. 2, three days. FIG. 7, 8, twelve days). - Elements of a bacterial culture of chard (Beta vulgaris, var. cicla) of twenty-four hours (FIG. 9, 10) and four days (FIG. 11, elements of the veil). - Elements of a bacterial culture of leek (Aillum porrum) of four days (FIG. 12 and twelve days (FIG. 13).

Pl. 234. - Cholera. FIG. 1 to 7, gr. 610: Penicilium form of cholera vibrio cultures (direct transformation, cholera cult. Pot).

Pl. 235. - Cholera. FIGS. 1 to 4, gr. 245. FIGS. 5 to 8, gr. 610: Penicilium form of cholera vibrio cultures (Bologna vibrio).

Pl. 236. - Cholera. FIG. 1, gr. 245. FIG. 2, gr. 100. FIGS. 3 to 9, 245: Vibrio de Bologne; Mucor form (FIG. 1, 2) - Vibrio de Jaritzin: Penicilium form (FIG. 3, 4); Sterigmatocystis form (FIG. 5); Aspergillus form (FIG. 6 to 9).;

Pl. 237. - Anthrax. FIG. 1, gr. 100. FIG. 2, gr. 245. FIG. 3, 4, 5, gr. 610: Gonidian forms of anthrax bacterial cultures. FIG. 1, 2: Aspergillus form. FIG. 3, 4, 5: Penicilium form.

Pl. 238. - Anthrax. - FIG. 1, gr. 245.-FIG. 2, gr. 610. FIG. 3, gr. 1100. Penicilium form of anthrax bacterial cultures.

Pl. 239. - Anthrax. - FIG. 1, 2, gr. 610. FIG. 3, gr. 1100.- FIG. 1: Germinative masses in a fourteen-hour-old anthrax broth culture. FIGS. 2, 3: Vegetation in broth of the spores of the Aspergillus form of anthrax cultures.

Pl. 240. - Plague. FIG. 1, 7, 8, gr. 245. FIG. 2 to 6, gr. 610: Penicilium form of plague bacterial cultures. - FIGS. 4 to 9: Direct transformation. FIG. 1, 2, 3: Passage through the guinea pig. FIG. 7, 8, 9: Marseille plague.

Pl. 241. - Plague. FIG. 1 to 7, gr. 610. - FIG. 8 to 13, gr. 1100, -FIG. 1 to 7: Penicilium form of a bacterial culture of Marseille plague. FIG. 8 to 12: Elements of bacterial cultures of the rat's organic mold. FIG. 8, 9, 12: Two-day-old culture of rat brain substance in broth. - FIG. 10, 11: Two-day-old culture of rat liver pulp in broth. - FIG. 13: Elements of a culture of Marseille plague on agar, after its passage through the guinea pig's body.

Pl. 242. - Penicilium form of the organic mold of the normal rat (Fig. 1 to 10, gr. 610).

Pl. 243. - Malta fever. FIG. 1 to 10, gr. 610: Penicilium form of bacterial cultures of Micrococcus melitensis. FIG. 11, 12, gr. 1100: Bacterial broth culture of orange organic mold.

Pl. 244. - Penicilium form of orange organic mold (Citrus aurantium) (FIG. 1 to 8, gr. 610).

Pl. 245. - Cerebrospinal meningitis. FIGS. 1 to 4, 6 to 8, gr. 245. FIGS. 5, 9, gr. 610: Penicilium form of meningococcus bacterial cultures.

Pl. 246. - Encephalitis lethargica. - FIGS. 1 to 17, gr. 610: Penicilium form obtained from the cerebrospinal fluid of patients with encephalitis lethargica.

Pl. 247. - Encephalitis lethargica. Gonococcus. - FIG. 1, gr. 245. -FIG. 2 to 8, gr. 610. FIG. 1 to 5: Penicilium form obtained from the cerebrospinal fluid of patients with encephalitis lethargica. FIG. 6, 7, 8: Penicilium form obtained by transformation of a gonococcus bacterial culture.

Pl. 248. - Encephalitis lethargica. - FIG. 1, 2, 3, gr. 245. FIG. 4 to 8, gr. 610: Aspergillus minimus and rudimentary Sterigmatocystis forms obtained from the cerebrospinal fluid of a patient.

Pl. 249. - Encephalitis lethargica. Aspergillus minimus form obtained directly by culture of the cerebrospinal fluid of a patient (FIG. 1, 2, gr. 245. - Fig. 3 to 6, gr. 610).

Pl. 250. Measles. -FIG. 1, 3, 4, 5, 7, gr. 245.-FIG. 6, gr. 100.-FIG. 8, gr. 610.-FIG. 1: Elements of the mold pathogen in the blood of patients. -FIG. 2: Elements of the bacterial culture of the pathogen in broth.-FIG. 3 to 5: Mold evolving into the Penicilium form, obtained by direct culture of blood.-FIG. 6: Elements contained in the veil of a culture of the pathogen.-Fig. 7, 8: Penicilium form of the pathogen in broth.

Pl. 251. Measles. -FIG. 1, 2, 4, 6, gr. 610. FIG. 3, 5, 7, 8, 9, gr. 245: Penicilium form of the measles pathogen. - FIG. 1, 2, 3: Measles No. 5. FIG. 4, 5: Measles No. 9. - FIG. 6 to 9: Measles No. 10.

Pl. 252. Measles. FIG. 1 to 10, gr. 610: Penicilium form of the measles pathogen. FIG. 1 to 5, 7 to 9: Measles No. 3. FIG. 6: Measles No. 10. FIG. 10: Measles No. 5.

Pl. 253. Penicilium form of the organic mold of lettuce (Lactuca sativa, var. capitata) (FIG. 1 to 9, gr. 610).

Pl. 254. Measles. -FIG. 1 to 11, gr. 1100: Elements of bacterial cultures of the measles pathogen. -FIG. 1 to 4: Germinal masses of the pathogen in the blood of patients. -FIG. 5: Seven-day and (FIG. 6) nine-day broth cultures of the pathogen (Measles No. 10). -FIG. 7 to 11: Elements of the veil of a broth culture (Measles No. 3).

Pl. 255. Elements of bacterial cultures of the lettuce mold (Lactuca sativa) (FIG. 1 to 9, gr. 1100). FIG. 1: Form of germ masses in a thirteen-day bacterial broth culture. - FIG. 2: Germ masses and the mycelial filaments they emit in a two-day broth culture. FIG. 3: Germ masses and bacillary elements in a thirteen-day broth culture. FIG. 5, 6: Twenty-four-hour broth culture. -FIG. 4, 6, 8, 9: Two-day broth culture.

Pl. 256. Scarlet fever. FIG. 1 to 5, gr. 245. FIG. 6, gr. 100.- FIG 2,3: Mycelium and fruiting bodies of the pathogenic mold in the patient's blood. -FIG. 5: Germinal masses and ball-shaped fruiting bodies in the venous blood. FIG. 6: Mycelial branch of the venous blood bearing, still adherent, several of its ball-shaped fruiting bodies. - FIG. 1, 3: Mold of a form identical to that of venous blood, developed on a carrot after inoculation with the patient's blood.

Pl. 257. Scarlet fever. FIG. 1, 3, 4, 5, 6, gr. 245. FIG. 2, gr. 610: Gonidian forms of the pathogenic agent. FIG. 1, 2: Verticilium form. FIG. 3: Aspergillus minimus, Sterigmato- cystis form. FIG. 4 to 6: Penicilium form.

Pl. 258. Scarlet fever. FIGS. 1 to 17, gr. 610: Penicilium form of the scarlet fever pathogen. FIGS. 1, 6, 7, 8, 11, 18: Scarlet fever No. 8. FIGS. 2, 3, 4, 5, 9, 12, 17: Scarlet fever No. 1. FIGS. 6, 7, 8: Scarlet fever No. 3. FIG. 14: Scarlet fever No. 5.

Pl. 259. Penicilium form of the organic mold of carrots (Daucus carota) (FIG. 1 to 15, gr. 610).

Pl. 260. FIGS. 1 to 14, gr. 610: Penicilium form of carrot organic mold. - FIGS. 15, 17, gr. 1100; FIG. 16, gr. 610: Sporothricum form. FIG. 18, gr. 610: Corymbisporium form.

Pl. 261. FIGS. 1 to 9, gr. 1100**:** Elements of bacterial cultures of the scarlet fever agent. FIGS. 1, 5, six days. FIGS. 2, 7, thirteen days. Elements of bacterial cultures of carrot organic mold. - FIG. 2, two days. FIGS. 6, 8, 9, six days. - FIG. 4,
seven days.

Pl. 262. - Chickenpox. FIG. 1, 4, 5, 6, 7, 10, gr. 610. -FIG. 2, 3, 8, 9, gr. 245: Gonidial forms of the chickenpox pathogen. FIG. 1, 6, 7, 8, 9, 10: Different types of the Penicilium form. FIG. 2, 4, 5: Sterigmatocystis form. -FIG. 3: Monostérigmate form.

Pl. 263. - Vaccinia. FIGS. 1 to 5, gr. 245. Gonidial mycelium of the vaccine mold. FIGS. 5, 6, gr. 610: For-FIG. 1, 2: Ovularia form. FIGS. 3, 4, 5: Verticilium form (Verticilium alboatrum). - FIG. 6, 7: Penicilium form.

Pl. 264. - Vaccinia. - Penicilium form of the vaccine mold (FIG. 1 to 16, gr. 610).

Pl. 265. - Vaccinia. Penicilium form of the vaccine mold: (FIG. 1 to 10, gr. 610).

Pl. 266. - Vaccinia. -FIG. 1 to 8, gr. 1100: Elements of bacterial cultures in broth of vaccine mold. FIG. 3: Culture of vaccine pulp in broth (twenty-four hours). FIG. 4, 5, 6: Same culture after six days, nine days and thirteen days. -FIG. 1, 2: Same culture (twenty-four hour broth) after its passage through the guinea pig. -FIG. 7, 8: Culture of glycerin vaccine in broth, two days and thirteen days old.

Pl. 267. Smallpox. Gonidian forms of the variola mold obtained directly by culturing smallpox scabs. FIG. 1, 2, gr. 245: Aspergillus form. FIG. 3, gr. 245; FIG. 4, gr. 610: Passalora form. FIG. 5, gr. 610; FIG. 6, gr. 245: Citromyces form. FIG. 7, gr. 245; FIG. 8, gr. 610: Spicaria form.

Pl. 268. Smallpox. Gonidial forms of the variola mold obtained indirectly (FIG. 1, 2, 4 to 7, gr. 610). FIG. 3, gr. 245: Peronospora form (Phytophtora infestans).

Pl. 269. Smallpox. - Gonidial forms of the variola mold obtained indirectly. FIG. 1, 2, gr. 245: Aspergillus form. - FIGS. 3 to 8: Penicilium form.

Pl. 270. Smallpox. Penicilium form of the variola mold (FIG. 1 to 11, gr. 610: Smallpox No. 1).

Pl. 271. Smallpox**.** Penicilium and Monosterigmata forms of the variola mold (smallpox No. 2). FIGS. 1 to 12, gr. 610: Penicilium form. FIGS. 13, 14, 15, gr. 610: Monosterigmata form.

Pl. 272. Penicilium form of the organic mold of potatoes (FIG. 1 to 12, gr. 610).

Pl. 273. Penicilium form of the organic mold of potatoes (FIG. 1 to 16, gr. 610).

Pl. 274. Smallpox. FIGS. 1 to 8, gr. 1100: Elements of bacterial cultures derived from the Aspergillus form of the variola mold in broth (FIG. 1, 2: 24-hour culture. - FIGS. 3, 4, two days. FIGS. 5 to 8, eight days).

Pl. 275. Foot-and-mouth disease. FIGS. 1, 3, 4, gr. 245. - FIGS. 2, 5, 6, gr. 610: Gonidian forms of the pathogenic mold. FIG. 1: Monosterigmata form. FIG. 2: Spicaria verticillata solani form. FIG. 3 to 6: Penicilium form.

Pl. 276. Foot-and-mouth disease. FIGS. 1 to 7, gr. 610: Penicilium form of the pathogenic mold. FIGS. 8, 9, 10, gr. 245: Verticilium alboatrum form. FIGS. 11 to 13, gr. 1100: Elements of bacterium broth cultures of the pathogenic mold that causes foot-and-mouth disease (FIG. 11, 12, twenty-four hours. -FIG. 13 ten days). -FIG. 14, gr. 1100: Bacterial elements contained in the serosity of hoof ulcers.

Pl. 277. Foot-and-mouth disease. Penicilium form of the pathogenic mold of foot-and-mouth disease (FIG. 1 to 17, gr. 610).

Pl. 278. Rabies. FIG. 1 to 4, gr. 245. FIG. 5 to 9, gr. 610: Gonidial forms of the rabies pathogen. FIG. 1: Aspergillus form. FIG. 2: Imperfect gonidial form, intermediate between the Mucor type and the Aspergillus type. FIG. 3: Cephalosporium acremonium form. -FIG. 4: Monosterigmata form. -FIG. 5: Corymbisporium form. -FIG. 6 to 9: Penicilium form.

Pl. 279. - Rabies. FIG. 1, gr. 245: Imperfect form related to the mold in Figure 2, Plate 278; chlamydospores. FIG. 2 to 16, gr. 610: Penicilium form of the rabies mold.

Pl. 280. Penicilium form of the organic mold of the dog (FIG. 1 to 15, gr. 610), obtained from the liver (FIG. 1 to 4, 6 to 12, 15), the lung (FIG. 5) and the spleen (FIG. 13, 14).

Pl. 281. Penicilium form of the dog's organ mold (continued), from the lung (FIG. 1, 5), liver (FIG. 2, 3, 6), and spleen (FIG. 4, 7). - Verticilium form obtained by culture of the pulp of the brain substance (FIG. 8, 9, 10).

Pl. 282. Penicilium form of the rabbit's organ mold. FIG. 1 to 16, gr. 610: Culture of spinal cord pulp.

Pl. 283. Syphilis. -FIG. 1, 2, gr. 610. FIG. 3, 4, gr. 245: Monosterigmata form of the pathogenic mold agcut of syphilis (grey mold); FIG. 4, gr. 930 and 6, gr. 610: Monosterig- mata form cultivated in colonies immersed in broth; spiral appendages of the mycelium (Treponema pallidum). - FIG. 6 (upper left corner): Bacillary forms of a culture of the Monosterigmata form on carrot.

Pl. 284. - Syphilis. FIG. 1, 2, gr. 1100: Grey-colored monosterigmatized form of syphilis. FIG. 3 to 7: White-colored monosterigmatized form of syphilis: FIG. 3, gr. 245; FIG. 4, 5, 6, 7, gr. 1100; FIG. 7: Spirillary appendages.

Pl. 285. Syphilis. Penicilium form of the agent of syphilis.-FIG. 1, 2, gr. 245; FIG. 3 to 8, gr. 610: Direct transformation of the monosterigmatized form.

Pl. 286. Syphilis. FIG. 1, 2, 3, gr. 100: Mucor form (Mucor Corymbifer) of the syphilis agent. FIG. 4 to 7, gr. 245. -FIG. 8, 9, 10, gr. 610: Penicilium form (directly obtained).

Pl. 287. Syphilis. - FIG. 1, gr. 245. FIG. 2, gr. 610: A type of the Penicilium form of the guinea pig organic mold, to be distinguished from the Penicillium form of the syphilis agent. - FIG. 3 to 8, gr. 245. Sterigmatocystis form of the syphilis agent. FIG. 4: Grey Monosterigmata form (indirectly obtained). - FIG. 5, 6, 7: Aspergillus minimus form. - FIG. 8: Aspergillus form.

Pl. 288. - Gonidian forms of the organic mold of the macaque monkey (Macacus cyno- molgus). FIGS. 1 to 3, gr. 610: Penicilium form. FIG. 4, gr. 60. - FIG. 5, gr. 91: Mucor cornutus form (origin: lung). FIGS. 6, 7, gr. 91. FIG. 8, gr. 60: Mucor corymbifer form (origin: brain).

Pl. 289 and 289 bis. Penicilium form of the organic mold of the macaque monkey (Pl. 289, FIGS. 1 to 16, gr. 610. - Pl. 289 bis, FIG. 1 to 15, gr. 610.

Pl. 290. Sporothricosis. Micrococcus melitensis**.** FIG. 1 to 3, gr. 610: Penicilium form of cultures of Micrococcus melitensis. FIG. 4, 6, 7, 8, gr. 610. -FIG. 5, gr. 245: Penicilium form of cultures of Sporothricum Beurmani obtained by direct transformation (FIG. 5, 7, 8) or by passage through guinea pigs (FIG. 4, 6).

Pl. 291. Sporothricosis. FIG. 1, gr. 245. FIG. 2, gr. 610: Penicilium form of Sporothricum Schenki cultures. -FIG. 3, 4, 9, gr. 245; FIG. 5, 6, gr. 610: Penicilium form of Sporothricum Gougeroti cultures (passage through the guinea pig).

Pl. 292. Icterohemorrhagic spirochetosis: Gonidial forms of the pathogen. FIG. 1, 2, 3, gr. 245: Aspergillus form. FIG. 4 to 9, gr. 610: Penicilium form.

Pl. 292 bis. -Penicilium form of the rat's organic mold (FIG. 1 to 9, gr. 610).

Pl. 293. Malaria. Elements of the pathogenic mold in the venous blood at the height of a fever (FIG. 1 to 4, gr. 1100. FIG. 5, gr. 175).

Pl. 294. Malaria. Monosterigmatized gonidial form of the pathogenic agent (FIG. 1, gr. 245. FIG. 2, gr. 610. FIG. 3 to 8, gr. 1100).

Pl. 295. Malaria. Gonidial forms of the pathogen. - FIG. 1, 2, gr. 245: Monilia and Stylosporium forms. FIG. 3, gr. 245: Monosterigmata form in development (spirillary appendages). FIG. 4, gr. 245: Aspergillus form. FIG. 5, 6, 7, gr. 245. FIG. 8, gr. 610: Penicilium form.

Pl. 296. - Sleeping sickness. Trypanosoma gambiense. FIG. 1, 2, gr. 1100.- FIG. 3 to 6, gr. 245. FIG. 1: Formation of trypanosomes by the germinal masses in the guinea pig inoculated with the spores of the Sporothricum form of the pathogen. FIG. 2: Flagellated spores (germinal filament) of the pathogen parasitizing a blood cell. - FIG. 3: Aspergillus form of the pathogen. FIG. 4: Sporothricum form (or Ovularia?) form. - FIG. 5, 6: Sterigmatocystis form.

Pl. 297. - Sleeping sickness. - Penicilium form of the pathogen (FIG. 1 to 7, gr. 610).

Pl. 298. Penicilium form of Lily of the Valley cultures. Lily of the Valley.-FIG. 1 to 16, gr. 610).

Pl. 299.- Trichophyton gypseum asteroides. FIG. 1, gr. 535. FIG. 2, 11, gr. 610. FIG. 3 to 10, gr. 245. FIG. 1: Monosterigmata form - or rudimentary Acremonium alternatum. FIGS. 2 to 11: Penicilium form.

Pl. 300. Trichophyton violaceum**,** - FIG. 1, gr. 245. - FIG. 2, 3, 4, gr. 610: Penicilium form.

Pl. 301. Trichophyton lacticolor. **-** FIG. 1 to 7, gr. 610: Pennicilium form.

Pl. 301 bis. Trichophyton crateriforme**.** - FIG. 1, gr. 175: Peronospora or Botrytis (Peronospora solani). - FIG. 2 to 7, gr. 610: Penicilium form.

Pl. 302. Trichophyton acuminatum. FIG. 1, 2, 4, 6, gr. 245. - FIG. 3, 5, gr. 610: Penicilium form.

Pl. 303. Achorion Schoenleinii. FIG. 1 to 4, gr. 245. -FIG. 5, 6, 7, gr. 610.- FIG. 1, 2: Evolved monosterigmat form. - FIG. 3 to 7: Penicilium form.

Pl. 304. Achorion gypseum**. -** FIG. 1 to 4, 6, 7, gr. 245; FIG. 5, 8 to 12, gr. 610. -FIG. 1 to 4: Normal fructifications of the Achorion. - FIG. 5 to 9: Evolution of the Achorion to the Penicilium type. - FIG. 10, 11, 12: Perfect Penicilium type.

Pl. 305.- Epidermophyton inguinale**.** FIG. 1, gr. 610. FIG. 2 to 8, gr. 245. FIG. 1, 2: Normal fructifications of the Epidermophyton. - FIG. 3 to 8: Penicilium form.

Pl. 306. - Brewer's yeast**.** FIG. 1, 2, 7, gr. 245. FIGS. 3 to 6, gr. 610: Penicilium form.

Pl. 307. Staphylococcus. FIG. 1, 3, 4, gr. 245. FIG. 2, 7, gr. 100. - FIG. 5, gr. 610. FIG. 1, 2: Aspergillus forms, and (FIG. 3 to 5) Penicilium from cultures of Staphylococcus pyogenes aureus. FIG. 6: Aspergillus form of Staphylococcus citrilis. FIG. 7: Aspergillus form of Staphylococcus blanch.

Pl. 308. FIG. 1, 2, gr. 245. FIG. 3, gr. 610: Pyocyanic bacillus. Penicilium form. FIG. 4, 6, gr. 245. FIG. 5, gr. 610: Bacillus subtilis. Penicilium form. FIG. 7, gr. 610. FIG. 8, gr. 245: Micrococcus catarrhalis. Penicilium form. - FIG. 9, gr. 245: Streptococcus. - Advanced monosterigmata form.

Pl. 309. Horse. - Penicilium form. (FIG. 1 to 16, gr. 610). Origin: FIGS. 1, 3, 6, 9, 10, 13, 16: liver. - FIG. 2, 4, 8: spleen. - FIG. 5, 7, 11, 12 muscle. FIG. 15: lung.

Pl. 310. Pig. - Penicilium form (FIG. 1 to 18, gr. 610). Origin: FIGS. 1 to 8, FIG. 12: liver. FIGS. 9 to 11, 13 to 18: lung.

Pl. 311. Sheep. Penicilium form (FIG. 1 to 14, gr. 610). - Origin: lung. Rhizopus form (FIG. 15, gr. 100).

Pl. 312. Guinea pig. Penicilium form (FIG. 1 to 13, gr. 610). Origin: FIG. 1, 2, 6, 8, 12, 13: liver. - FIG. 3, 4, 5, 7, 10, 11: spleen. - FIG. 9: lung.

Pl. 313. Guinea pig. Penicilium form (FIG. 1 to 11, gr. 610). Origin: FIG. 1, 7, 8: lung; FIG. 2 to 6, 9 to 11: spleen.

Pl. 314. Guinea pig. Citromyces form (FIG. 1, 2, gr. 245, FIG. 3, gr. 610). Origin: lung. Penicilium form (FIG. 4 to 11, gr. 610). Origin: lymph nodes.

Pl. 315. - Mouse. - Penicilium form (FIG. 1 to 15, gr. 610). FIGS. 7, 10 to 12: liver. Origin: FIGS. 1 to 6, 8, 9, 13, 14, 15: heart. Sporothricum form (FIG. 16, gr. 610). Origin: brain.

Pl. 316. - Peacock (Pavo cristatus). **-** Penicilium form (FIG. 1 to 19, gr. 610). Origin: lung.

Pl. 317. - Sultana fowl (Porphyrio Edwardsii). Penicilium form (FIG. 1 to 14, gr. 610). Origin: spleen. - Aspergillus form (Fig. 15, gr. 100). Origin: lung.

Pl. 318. Grey parrot (Psittacus cinnah)**.** - Penicilium Form (FIG. 1 to 10, gr. 610). Origin: lung. - Aspergillus minimus Form (FIG. 11 to 13, gr. 245). Origin: heart.

Pl. 319. - Turtle (Testudo ibera)**.** Penicilium Form (FIG. 1 to 11, gr. 610).

Pl. 320. Sayman (Alligator mississipiensis). - FIG. 1, gr. 100. FIG. 2, gr. 245. FIG. 3, gr. 610: Verticilium Form. FIG. 4, 5, 6, gr. 245: Aspergillus Form. FIG. 7 to 11, gr. 610: Penicilium Form.

Pl. 321. Frog (Rana temporaria). -FIGS. 2 to 14, gr. 610: Penicilium form. -FIG. 1: Form from the agar culture. - FIGS. 15, 16, gr. 245: Mucor form.

Pl. 322. - Eel. (Anguilla anguilla). - FIGS. 1 to 11, gr. 610: Penicilium form.

Pl. 323. Whiting (Merlangus merlangus) - FIGS. 1 to 7, gr. 610: Penicilium form. FIG. 8, gr. 245: Fusarium form. FIG. 9, gr 245: Aspergillus form. - FIG. 10, gr. 245. FIG. 11, gr. 1100: Mold obtained from muscle tissue; fasciculated arrangement of very long mycelial branches.

Pl. 324. - Black salsify. - Scorzonera hispanica**.** Penicilium form (FIG. 1 to 15, gr. 610).

Pl. 325. Eucalyptus globulus**.** FIG. 1 to 11, gr. 610: Penicilium form. Origin: Floral receptacle.

Pl. 326. Amanita phalloides**.** FIG. 1 to 15, gr. 610: Penicilium form. FIG. 16, gr. 245: Fusophragmium form.

Pl. 327. Amanita citrina**.** - FIGS. 1 to 11, gr. 610: Penicilium form.

Pl. 328. Collybia velutipes. - FIGS. 1 to 12, gr. 610: Penicilium form.

Pl. 329. Lycoperdon pratense**.** FIGS. 1 to 15, gr. 610: Penicilium form. - FIG. 16, gr. 245: Monosterigmata form.

* * *

Docteur J. TISSOT
Professeur honoraire de Physiologie Générale
au Muséum National d'Histoire Naturelle

◆

LA CATASTROPHE
DES
VACCINATIONS OBLIGATOIRES

◆

**Elles sont très dangereuses,
peuvent inoculer une diphtérie grave
et d'autres maladies
à vos enfants
qu'elles tuent par milliers.**

◆

DÉFENDEZ LES

Laboratoire annexe de Physiologie générale
Professeur J. TISSOT, 42, boulevard Saint-Marcel, Paris (5e)

Doctor J. TISSOT
Honorary Professor of General Physiology at
the National Museum of Natural History

◆

THE CATASTROPHE OF COMPULSORY VACCINATIONS

◆

They are very dangerous and can
inoculate your children with serious
diphtheria and other diseases,
killing them by
the thousands.

◆

DEFEND THEM

Annex Laboratory of General Physiology,
Professor J. TISSOT, 42, boulevard Saint-Marcel, Paris (5)

THE CATASTROPHE OF MANDATORY VACCINATIONS

On June 25, 1938, the Senate, deceived by false statements, passed an odious law that deprived the French of the right to freely dispose of their bodies, as recognized by the Declaration of the Rights of Man in 1789, and of the inalienable right to protect the health of their children.

The result of this odious law has been that the diphtheria vaccine has caused a terrible catastrophe since 1940, inoculating approximately 150,000 children with severe diphtheria and murdering more than 15,000, not to mention a chronic illness created by the vaccine in a considerable number of children; and, monstrously, all this so that the vaccine manufacturer could make enormous profits.

Thus, we take no more notice of the lives of your children than we do of that of a guinea pig in a medical laboratory.

This dreadful catastrophe and the fact that the public was thus defenseless by the law of June 25, 1938, to the machinations of the vaccine manufacturer determined Doctor J. Tissot, Honorary Professor at the National Museum of Natural History, to take on this defense.

To this end, he had a brochure printed entitled: The Catastrophe of Compulsory Vaccinations. It is on sale throughout France at newsagents and bookstores. Ask for it and make it known to those around you. It will show you the means to protect yourself against the dangers of vaccines and against diphtheria itself without using any vaccine.

It will prove to you that the only way to protect yourself against the catastrophic effects of vaccines is the rapid formation of a national league called "Health and Liberty," whose goal is the defense of the liberty and health of the French against all causes that could be harmful to them.

For this league to be powerful enough, it must have at least two million members. All families must join, even the father and mother if possible, as well as single people, because everyone is now threatened by vaccines.

This league will defend you energetically and pursue the abolition of the shameful, dishonoring obligation to submit yourself and your children to inoculation with deadly vaccines, solely to ensure the financial prosperity of a vaccine factory.

You and your friends, send your membership without delay, because new bills threaten you all, even adults.

To ensure the functioning of the league and its means of action, an annual subscription of at least 50 francs is essential. It will be reduced subsequently, as the number of members increases and as soon as possible.

We warn you that as the action of the Health and Liberty League develops, the reaction of the vaccine manufacturer will intensify in various forms, including intense publicity boasting.

PREFACE

Great concern and strong opposition have long been manifested among the public and throughout France against vaccinations due to the accidents they have caused. Despite this, the public is still generally unaware of the appalling catastrophe they have caused, which is being hidden from them and about which there is complete silence in the medical and even political press.

When the reader has fully read this brochure, they will certainly realize that vaccinations have neither the protection of public health nor the progress of hygiene as their goal nor their result; the French only play the role of guinea pigs, subjected to the sale of vaccines to their detriment in order to ensure the financial prosperity of a commercial enterprise.

The vaccine trade has become an extremely serious danger for the public, which currently has no means of defending itself, having been delivered to its mercy by an odious law, that of June 25, 1938, which, by making vaccinations compulsory, deprived the French of the most essential and inviolable of rights recognized by the 1789 Declaration of the Rights of Man: the right to freely dispose of one's body, a right which entails, for each person, the right to protect the health of one's children.

The result of this odious law has been, since 1941, the inoculation, by vaccine, of serious diphtheria in approximately 150,000 children and the murder of more than 15,000 others.

The public will learn later that, if there had been the slightest concern for its health, the imperative order would have been given, in 1925, to cease the diphtheria vaccination. I wrote this brochure especially for the public, to make them aware of the ineffectiveness and dangers of the most commonly practiced vaccinations, and above all to educate them on their rights and the most effective means to employ, either to ensure their own defense or to avoid diphtheria and other so-called contagious diseases without using any vaccine or chemical product.

The following examination will focus successively on:

1. Diphtheria vaccination.
2. Tetanus serum and vaccination.
3. Typhoid vaccination.
4. Tuberculosis vaccination with BCG.
5. Rabies treatment and the false Pasteurian dogma of immunity created by inoculation with an attenuated virus.
6. Smallpox vaccination.

Diphtheria Vaccination

An odious law was passed on June 25, 1938, depriving the French of the most essential right recognized by the 1789 Declaration of the Rights of Man: the right to freely dispose of their persons, which entails the inviolable right of parents to protect the health of their children. This odious law forces them to allow their children to be inoculated, under threat of sanctions, with a series of dangerous vaccines.

Was there a motive of capital importance for committing such a serious attack on the most essential human right? No! The members of Parliament passed this law because they were odiously deceived. Was the intention behind this law to bene- fit the French? No! It was only to enormously increase the sale of the vaccine through the compulsory and repeated vaccination of millions of children. Here are the facts**:**

Diphtheria vaccination with toxoid began in 1923. From the outset, it caused numerous accidents. These were ignored.

In 1925, when an epidemic broke out in the Army of the Rhine, it was decided to carry out vaccinations. Military doctor Zæller conducted a first control test in Mainz. He vaccinated 305 recruits with two injections of diphtheria toxoid given three weeks apart. Other unvaccinated recruits served as controls.

Eleven cases of diphtheria occurred among the 305 vaccinated during the three weeks separating the two injections: only one case occurred among approximately 700 unvaccinated recruits. The conclusion was therefore that vaccination had the unexpected, but definite, effect of causing eleven times more cases of diphtheria among the vaccinated than among the unvaccinated. This truly catastrophic result of toxoid therefore imperatively required its abandonment.

But this was not the case, because such a decision would have eliminated the sale of this vaccine, which had been inaugurated two years earlier. Also, it was claimed that the cases of diphtheria among the vaccinated had occurred in incompletely immunized men, that is, too recently vaccinated, and that they should be removed from the group of

vaccinated people and included in the group of unvaccinated people, who, however, were even less immunized, since they were not vaccinated at all.

It will certainly not escape the reader that this reasoning is contrary to common sense, that it is false. This distortion of the truth therefore had the effect of completely changing the result of Zæller's experiment, which, as a result, was transformed into less than one case among the vaccinated for eleven cases among the unvaccinated. The vaccine was thus absolved of its crime and could continue to be marketed, continuing its misdeeds in practice as well as its fruitful relationship with its manufacturer. Never, over the centuries, has one seen, in science, such a distortion of a statistic and, for such a barely disguised purpose, such an absurd mystification which is an outrage to the common sense of the public, who will surely not be fooled. Zæller's experiment proved, denounced the catastrophic action of diphtheria toxoid and imperatively demanded its abandonment.

It was therefore easy to avoid this catastrophe and it should never have happened. The cause that provoked it was therefore: The distortion of Zæller's experiment in order not to hinder the vaccine trade.

* * *

The facts I have just set out, perfectly well known, historically proven, one might say, took place in 1925.

On December 6, 1927, the Academy of Medicine voted the following resolution:

<< *The Academy of Medicine, considering that diphtheria manifested itself last year with a special malignancy leading to a high mortality rate in our country; that anti-diphtheria vaccination has already been used in France and in several nations;*

<< *That this vaccination has proven its effectiveness and its harmlessness*;

<< *Requests the public authorities that this method be systematically instituted among children, particularly among those who attend schools... etc.* (1)."

Thus, two years after Zæller's experiment proving the catastrophic action of toxoid, the Academy of Medicine still affirmed its effectiveness and safety, which it had not verified itself and for which it had not demanded the slightest proof from the manufacturer.

It was under such conditions that the right to forcibly inoculate young soldiers with this vaccine was first obtained.

(1) *Chamber of Deputies. Session of 1930. Appendix to the minutes of the 2nd session of July 11, 1930.*

From 1927 to 1938, toxoid caused a considerable number of accidents, some resulting in death (1). It was noticed that vaccinated people frequently contracted diphtheria more serious than unvaccinated people: there were protests from many doctors. This was ignored.

On the contrary, a bill was presented to Parliament making this vaccination compulsory, with the obvious aim of enormously increasing sales of the vaccine.

Indeed, the report on the law, read to the members of the Senate, contains the following information:

"*The expenses will certainly be offset by the elimination of 2,000 annual deaths and the treatment, each year, of 20,000 cases of diphtheria... The vaccination costs will certainly be less than the savings they will bring about by the elimination of diphtheria*" (2).

As we will see, this information, manifestly false, exactly contrary to the truth, has odiously misled the members of the Senate; it was only because their opinion was distorted that they voted for the law. Indeed:

If we examine the number of diphtheria cases from 1912 to 1945, we see that, during optional vaccination, from 1923 to 1940, the annual number of diphtheria cases was never lower than the number of cases before vaccination; that, during the first seven years of optional vaccination, from 1923 to 1930, vaccinations increased the annual number of cases from 12,000 to 24,000.

These facts already demonstrated the falsity of the statements made in the Senate. Moreover, this falsity was proven, in 1938, by the catastrophic results of vaccinations in Germany, where they caused the number of diphtheria cases to increase from 30,000 in 1926 to 147,000 in 1937.

This enormous increase, well known in 1938, therefore formally opposed any claim or belief that the new law would eliminate diphtheria in France.

This new outrage against the truth therefore shows that it was only by mislea- ding the opinion of the members of Parliament that this odious law could be passed, depriving the French of the most precious of freedoms, that of freely disposing of their bodies. These false assertions are written in the Official Journal of June 28, 1938, page 7371, and the application of the odious law has given them a terrible response in the form of 150,000 cases of diphtheria inoculated by the vaccine and more than 15,000 children murdered by it since 1941. And here are the results of the application of this odious law, according to official statistics:

(1) *They are set out in the book by Doctor P. Chavanon, Nous, les cobayes... (Editions Médicis, Paris.)*
(2) *Official Journal of June 25, 1938, page 7371.*

The number of diphtheria cases, which was 13,795 in 1940, rose to 46,750 in 1943, to 41,500 in 1944, and to 45,500 in 1945, and the mortality rate was 2 to 4 times higher for these cases of diphtheria than for those of unvaccinated people. Including the year 1946, there were, in total, approximately 150,000 cases of diphtheria from 1940 to 1946 in addition to the normal number of cases before vaccinations, and more than 15,000 children killed, or rather murdered, by toxoid Depending on the department, the number of cases was 2 to 5 times, 10 times, 15, 20, 25 and up to 35 times greater than before vaccinations, a fact which proves that each department was the site of an experiment whose result confirmed that of Zæller or was even more demonstrative.

The conclusions of these facts are the following:

1. The verification of the safety of the vaccine, imposed by law, was never carried out since it causes diphtheria and has killed thousands of children;

2. The statement by the Academy of Medicine, dated December 6, 1927, "*that this vaccination has proven its safety*" was therefore inaccurate and seriously distorted the opinion of the members of Parliament; it therefore seriously contributed to the passage of the 1938 law and consequently to the provocation of the catastrophe.

* * *

It has been claimed that the diphtheria that develops in vaccinated people (called post-vaccinal) after the injection of toxoid is due to a decrease in resistance to the natural infection caused by the vaccine. This reason is not accurate, because it is equally valid for all viruses, and vaccinated people do not contract measles, scarlet fever, or chicken-pox, but only diphtheria.

Furthermore, statistics have shown that during epidemics, the annual number of cases has never exceeded double the number observed in ordinary years. However, the number of cases observed among vaccinated people has been 3, 5, 10, 15, 20, 25, and up to 35 times greater than among unvaccinated people, depending on the department.

Clearly, these cases of diphtheria are therefore not due to natural contagion, but to an exceptional, artificial, and induced cause, which is the contamination of the tonsils by the vaccine itself.

Proof of this contamination is provided by diphtheria that develops 12 to 24 and 48 hours after the vaccine injection. If, from this time, we deduct the time needed for the virus to evolve from the microbial state to the false diphtheria membrane state, which is at least 12 to 48 hours since the virus is attenuated, we see that the infection occurs immediately after the vaccine injection.

Another proof, and this is the one that is directly the constant cause of the infection, is the continuous passage of the granular elements of the viruses through all the glands of the body and which are thus carried away in the fluids they secrete. This is a normal, physiological, and inevitable phenomenon. It is through this that the saliva of a rabid dog becomes virulent and gives rabies to those it bites, and that Malta fever is contracted by drinking the milk of goats contaminated by this disease. Finally, further proof is provided by the fact that diphtheria in vaccinated people is always more serious and has a mortality rate 2 to 4 times higher than that of unvaccinated people.

It is therefore the toxoid itself that inoculates diphtheria.

* * *

Everything set out in this brochure should persuade the public that the use of vaccines has nothing to do with the protection of their health or with progress in hygiene. Only a commercial question is at stake, and until now, the public has strictly played the role of guinea pigs used solely to ensure the financial prospe- rity of the vaccine trade; from this role, it has reaped, since the beginning of vaccinations, only 200,000 cases of serious diphtheria inoculated into children, more than 20,000 others murdered, and, in addition, a large number of cases of paralysis, nephritis, and other damage.

Now they are trying to shift the question by alleging that, in an epidemic, only the unvaccinated contract diphtheria, based on statistics whose value we know. This is inaccurate. But, even if it were true, it would only mean that, to achieve this, it would always be necessary, beforehand, to over-vaccinate and inoculate more than 200,000 children with diphtheria and to murder at least 20,000 of them. Moreover, since immunity only lasts about five years, this catastrophe will have to be repeated twice more, at the ages of six and eleven, otherwise the children will become susceptible from the age of six.

One would have to be mad to dare to support such reasoning. The public will obviously realize that through incapacity, lack of foresight, criminal imprudence, and financial greed, it has been drawn into a terrible catastrophe from which it can only escape by refusing vaccinations, repealing the law of June 25, 1938, and opening an investigation.

The public must become aware of the current dramatic situation through the following reasoning:

The result of compulsory vaccinations was that, in 1943 alone, there were 46,750 cases of diphtheria instead of 13,795 in 1940, or about 33,000 cases of severe diphtheria inoculated by the vaccine in children and at least 3,300 of them murdered by it, the mortality rate being 10 to 13 per 100 cases. Such a catastrophe would have required stopping vaccinations and conducting an immediate investigation if there had been, at the head of the Ministry of Public Health, a competent minister aware of his duties and responsibilities.

Why then was the catastrophe allowed to continue? It's because the vaccine trade has monopolized all the machinery and authority of this ministry; it reigns supreme, makes decisions that are useful to it, and prevents any that could be harmful. Some of the ministry's officials even come to broadcast propaganda for the products of this trade.

However, stopping vaccinations in 1943 would have been to implicitly acknowledge the serious errors that caused the catastrophe and raise the question of responsibility. This is why we do not want this solution at any price, why we oppose it, and why we will continue to inoculate children with diphtheria and murder them by the thousands as long as your will does not oppose it.

This is why you have no help to expect, neither from the Ministry of Health, nor from the Academies and Learned Societies. You have only yourself to rely on. Your enemy, the vaccine trade, is as powerful as it is dangerous. But you will realize that you have an irresistible power at your disposal: the truth and your rights. Here's how**:** It is diphtheria toxoid that inoculates your children with diphtheria and kills them by the thousands. It follows that the law of June 25, 1938, must be repealed without delay because:

1. His vote was obtained by misleading members of Parliament with assertions proven false by the current catastrophe;

2. Because the safety of the vaccine imposed by law has not been realized;

3. Because its result was rigorously contrary to the legislator's intention;

4. Because, by removing the most essential of the rights recognized by the Declaration of the Rights of Man of 1789, that of freely disposing of one's body, it is unconstitutional;

5. Because the vaccinations, it made compulsory, are a disaster, a catastrophe for the child population.

The law requires children and soldiers to receive under the skin vaccines that protect against disease and are harmless, but not vaccines that cause it and kill. Therefore, the current catastrophe puts you in a state of self-defense against vaccinations and gives you the right to refuse them without any sanctions being taken against you.

You have three ways to defend yourself:

1. Use your right of self-defense by refusing all vaccinations. You have a formidable weapon there:

2. Get all your elected officials to act; protest in groups and demand the repeal of the law of June 25, 1938;

3. Group yourselves in every city, in every factory, in every department into an anti-vaccination league that will be baptized everywhere with the same name of Health and

Liberty. All the leagues will quickly come together into a powerful federation that will impose its will.

Let all those who have had a child murdered by vaccines, or paralyzed or made sick, take the initiative of forming a defense group to form a league.

Let all those who want to defend children and prevent their continued murder for the sole benefit of the vaccine trade stand up and get to work!

Everyone distribute this brochure and make it known!

* * *

Vaccines cause disease and kill your children; But there is a sure way to avoid it without using any vaccine. The way is to know where the virus is, and knowing where it is, to destroy it before ingesting it, because it is contained in our food. I already made this me thod known as early as 1926, in the work Constitution of Animal and Plant Organisms; Causes of the Diseases that Affect Them (see page 503, 1st vol., and page 18, 3rd vol.). But it was stifled, it was prevented by bans on medical and scientific journals, from being known and put into practice because it suppressed the trade in catastrophic vaccines; here it is:

Diphtheria viruses are numerous and contained in cereal flours and in cow's milk, often contaminated by bits of manure from the cereal straw bedding that fall into the bucket during milking. The croup virus is the spore of Cladosporium herbarum, a common disease of cereals.

By boiling milk and foods containing cereal flours for 20 minutes, you will prevent your children from diphtheria, childhood poliomyelitis, measles, and scarlet fever. Before boiling the milk, add a sufficient quantity of water to prevent it from reducing. Since cereal porridges do not lend themselves well to sterilization, it is advantageous to replace them, when feeding children, with vegetable porridges, potatoes, leeks, carrots, with added pea flour, lentil flour, and rice grains, boiled for at least 20 minutes, which have the advantage of not containing the viruses of the diseases designated above.

Antitetanus Serum and Vaccine

Experiments conducted in 1890 appeared to demonstrate that, when an animal is inoculated with tetanus, its blood serum, injected into another animal, prevents the same inoculation from developing tetanus. This conclusion was false and inapplicable to humans because it had been insufficiently studied.

Although falsely interpreted, this conclusion served as a pretext for the manufacture of an antitetanus horse serum, which was immediately put on the market with the false claim that it protected against tetanus when injected after an injury.

However, it was already known, even before this time, that a patient cured of tetanus is never immune to a second attack, a fact which means that no serum can protect humans against their spontaneous tetanus, and that the commercially available serum is completely devoid of this power.

In the third volume of the work indicated on page 7, I have during a long study, provided superabundant, formal proof of this fact and demonstrated that, moreover, it confers on all those into whom it is injected, colibacillosis of the horse that is kept for decades, as well as the very dangerous state of anaphylaxis in the case of a second injection of serum, even given ten years later. Thus, the guarantee of the effectiveness of this serum against tetanus is false and its sale a gigantic deception that has lasted throughout the world for more than fifty years. The same criticism is leveled at the tetanus vaccine, which is incapable of vaccinating against tetanus and confers colibacillosis.

What will people think abroad of French science when they learn that the Academy of Medicine voted in 1947 to generalize this deception of tetanus vaccination?

Conclusion. Never allow yourself to be injected with either tetanus serum or tetanus vaccine: they have no protective effect and only give you colibacillosis.

Anti-typhoid vaccination

The triple vaccine injection (T. A. B.) often causes a very strong reaction and it is not uncommon for it to affect the health of the vaccinated person for several years. Although rarely, it can cause sudden death.

Immunization with this vaccine is doubtful because, six months after recovering from typhoid fever, one can contract a second. Rathery and Mathieu noted that a large number of soldiers contract typhoid fever less than a year after vaccination. Dr. Jouve Balmelle noted that, in his hospital service, out of 100 soldiers with typhoid, an average of 35 were vaccinated. Recently, an epidemic broke out in the American army, in Yokohama, where 24 American soldiers contracted typhoid fever, although they had been vaccinated and revaccinated.

A newspaper note (Le Monde, February 11, 1948) informed us that the High Council of Hygiene has just prepared a nice little law to make anti-typhoid vaccination compulsory, which would realize the golden dream of bringing more than a billion into the vaccine trade coffers (30 million subjects at a profit of 50 francs each).

Only this coffers would gain from vaccinations because they have a much more harmful effect than natural infection.

Indeed: it has been admitted that typhus vaccination caused 4 or 5 deaths annually from 1936 to 1939 out of 200,000 troops, or 1,000 out of 40 million during the days following it, not including the deaths of vaccinated people who die from it during the year, particularly from typhoid.

Now, for 4,000 annual cases of typhoid infections per 40 million inhabitants from 1936 to 1939, there would be 200 to 400 deaths with a mortality rate of 5 to 10 percent, or 1 to 2 per 200,000 inhabitants, while vaccination caused 4 or 5 or more in the army per 200,000 men and 1,000 per 40 million.

This vaccination also causes numerous cases of nephritis, liver diseases, etc., which continue to develop after the men return home and for which the number of deaths remains unknown.

Out of 200,000 inhabitants, natural typhoid kills only 1 or 2; vaccinations kill more than 5 and sicken thousands. Only the vaccine manufacturer benefits from the vaccination. The vaccinated loses.

Due to the short duration of the weak protection conferred, it would be necessary to repeat the vaccination and expose the inhabitants to its risks and accidents every 10 or 12 months. To consider such a possibility denotes total recklessness.

As a result of these facts and considerations:

It would be absurd, monstrous, to prevent typhoid fever in a single inhabitant, to inject the three T.A.B. vaccines into 10,000 or 20,000 others, in whom they would cause more deaths and accidents than natural infection and to whom they would confer the three chronic typhoid infections with their dangerous mycoses.

Is BCG a vaccine and is it capable of immunizing humans against tuberculosis?

The nature of tuberculosis, as indicated by Villemin, then by Robert Koch upon discovering his bacillus, will have been one of the most serious errors of the 19th century.

The tuberculosis bacillus is not a microbe in the absolute sense of the word, a microbe like those of diphtheria or typhoid fever. It is a small microscopic body, with a shape very different from that of a microbe. It has exactly the shape of the dumbbell used for physical exercises; it is made up of a rod with a ball at each end; by assembling with others by its balls, it forms either networks or filaments which constitute the tuberculous tissue and also the entire body and organs of all living beings, animals and plants.

Its shape is unique and it is universal. In a word, it constitutes the living matter of every being, the brain, nerves, muscles, lungs, liver, etc.

It is by undergoing a denaturation or degeneration in its properties, while retaining its dumbbell shape, that this small body becomes a tubercular Koch bacillus, that is, it acquires the harmful property of producing tuberculous tissue that replaces the healthy tissue it destroys. Having acquired this property, this universal dumbbell first constitutes long filaments that insinuate themselves into the healthy tissue of the lung, for example, and gradually destroy it until they form the cavities called caverns.

Thus, it is in the patient himself that this degeneration takes place; consequently, tuberculosis is autogenous, that is, it arises spontaneously in the tubercular patient and not because he is infected by Koch's bacilli coming from outside, that is, by contagion.

Attempts are being made to cover up these discoveries. Why? Because they demonstrate the enormous errors of BCG, the ox tuberculosis bacillus that Calmette and Guérin cultivated and re-cultured many times on a medium containing ox bile and which, as a result, eventually lost the ability to develop tuberculosis when inoculated into oxen.

If they concluded that BCG could vaccinate, it was because they were ignorant of the nature and origin of Koch's bacillus, and:

1. That the bovine tubercle bacillus is the degenerated constructor dumbbell of the ox;

2. That the loss of the tuberculizing power of this dumbbell, in the presence of ox bile, had only returned it to the state of a normal ox dumbbell, of which the latter normally possesses an enormous quantity that does not immunize it and in no way prevents it from becoming tubercular;

3. That it is contrary to common sense to claim to immunize against or with the constructor dumbbell, a normal constituent of tissues;

4. That claiming to immunize against the human tuberculosis bacillus with the bovine tuberculosis dumbbell, which is as specifically different from it as a man differs from an ox, is a serious error of principle;

5. That since tuberculosis is an autogenous disease, any immunization against it is impossible, autogenous diseases never immunizing the patient. Example: tetanus, cancer.

In summary:

BCG cannot and will never possess any immunizing power due to its nature. It is and will always remain the constructive dumbbell of the ox once it has returned to normal, of which this animal possesses a large quantity that does not prevent it from becoming tubercular; any progress is impossible for it.

To conclude, I will add that the BCG dumbbell constructor of the ox, being specifically different from that of man, is therefore heterogeneous and virulent for the latter, a fact which explains the numerous accidents that its application has caused and which have caused its use to be abandoned in France and in many foreign countries. Its use could only expose children to the return of its degeneration and to the development of bovine tuberculosis, the type of which is now known and which V. Simon has called: Calmette-Guérin tuberculosis.

Conclusion. - *Refuse vigorously and prohibit in writing the inoculation of your children with BCG. They can only reap a harmful effect and in no case a favorable one.*

Pasteur's anti-rabies treatment

The Pasteurian dogma of immunity created by the inoculation of an attenuated virus is false.

Any disease caused by a foreign microbe has two phases: an acute phase with fever, where the microbe multiplies for 8 to 15 or 20 days, including the incubation and the period of illness, and a chronic phase that can last until death and includes the most serious symptoms of the disease, some of which occur at the very beginning of this period, others much later.

To multiply in the blood, the microbe itself produces a substance called antitoxin, which clumps its elements together and causes them to change into the mold state that characterizes the beginning of the chronic period. This mold will lodge in the organs where it causes disorders (paralysis, nephritis, etc.) while continuing to form the antitoxin. This is the immunizing body, but it does not protect against the disorders caused by the mold, since it is the mold that gives rise to it. The principle of immunity is therefore false. Moreover, it is false:

1. Because the supposedly immunizing antitoxin is produced by the virus and ensures its evolution and not its destruction;

2. Because a virus, having become a vaccine through attenuation, can recover its original virulence and cause the disease with all its severity. *The current catastrophe is due to this fact;*

3. Because the vaccine inoculates all vaccinated people (100 percent) with chronic disease, that is, permanent infection of the virus with the risk of all its serious accidents, for example, paralysis and nephritis for the diphtheria vaccine;

4. Because, practically, vaccinations against typhoid fever, smallpox, and diphtheria do not prevent contracting the disease six months or a year later.

* * *

The problem of rabies has never been clearly posed. The reader has just seen that any disease caused by a foreign microbe has two phases: an acute phase of microbial multiplication that lasts 8 to 15 or 20 days, and a chronic phase, of a mycelial nature, which begins from the 10th to the 20th day and includes the most serious symptoms of the disease.

However, until now, the rabies crisis has been considered as marking the period of rabies and the end of its incubation period, whereas it is an incident of its chronic phase, which explains why it can appear as easily 20 days after the bite of the rabid dog as six months later. This distinction is of capital importance because we saw, on the previous page, that we cannot immunize against the symptoms of the chronic phase of diseases because they are due to lesions caused by the virulent mold, which, at the same time, forms the antitoxin, the supposed cause of immunity.

It is therefore the virulent mold that simultaneously causes immunity and the damage that we claim to avoid by inoculating a vaccine. This is why the rabies crisis, an accident of the chronic phase, could not be avoided by Pasteur's so-called rabies marrow vaccine.

The rabies virus is a mold of the genus Aspergillus that I isolated. The rabies crisis only occurs when the mold develops in the brain, and it only develops there, after a bite, in one in six to ten or fifteen individuals.

This is why it is inconsistent, but those bitten by a rabid dog, and those vaccinated who are not, are still infected by the rabies virus and suffer the other disorders associated with it.

Pasteur made a serious medical error by claiming to stop the progression of an ongoing disease with a second inoculation of his virus. But he made another, much more serious one by forgetting that the street rabies virus from biting dogs was sufficient on its own to immunize those bitten, better and faster than his rabbit rabies marrow vaccine, the injection of which was therefore not only useless but had the serious disadvantage of inoculating the vaccinated with rabies from the fixed rabbit virus, particularly those who, bitten by a non-rabid dog, were not at risk of contracting it.

The anti-rabies treatment initially appeared effective and harmless, but the deaths of the Pelletier and Bonenfant children opened the black series at the end of 1885.

Pasteur having made the process more intensive at the end of September 1886, it became disastrous and, in less than three months, he inoculated 11 people with paralytic rabies who died from it. In 1885, there were 22 cases of rabies in France among the vaccinated and 17 among the untreated, or 39 in total, instead of an annual average of 30.

On January 11 and 18, 1887, Professor Peter demonstrated at the Academy of Medicine that anti-rabies treatment did not cure street rabies, but did inoculate that of the fixed rabbit virus.

Thus, this famous discovery, published on October 27, 1885, the day that the president of the Academy, Bergeron, declared *"the most memorable date in science,"* was only one of the three most enormous errors that medical science would experience over the centuries due to the medical and biological incompetence and ignorance of one man.

It is this monstrous, absurd, criminal treatment which is the main claim to fame of Pasteur because senseless, charlatan propaganda presented him to universal admiration as the greatest discovery of modern times. Neither Professor Peter's demonstration nor the victims of this criminal procedure could prevent it from continuing for sixty years. It was preferred to inoculate rabbit rabies into all the poor bitten victims who come, trusting, to ask for help because to stop it would have been to recognize Pasteur's error and destroy a fragile glory that is urgently needed to support the failing reputation of catastrophic vaccines. The public will judge.

It must be known that no scientific means currently allow the progression of rabies to be stopped and that any vaccine used for this purpose can only confer it with certainty to those into whom it is injected.

Smallpox Vaccination

The Jennerian vaccinia virus, very similar to but different from the smallpox virus, does not vaccinate against smallpox. This fact is clearly established by smallpox epidemics that also affect vaccinated people. Here is proof:

Despite almost complete vaccination of the inhabitants of the city of Leicester (England), the 1872 epidemic caused so much smallpox among the vaccinated that they abandoned vaccination and successfully replaced it with prophylactic measures.

The smallpox epidemic of 1918-1919 in the Philippines, whose nation has been, since 1905, one of the most completely vaccinated in the world, struck both vaccinated and unvaccinated people indiscriminately, causing 145,317 cases and 63,434 deaths. Smallpox mortality reached 65.3% in Manila, the most highly vaccinated city in the country, compared to only 10% before 1905 **(1)**.

The same facts have been observed in Germany and Japan. Doctor R. Halle Backenwelle, general vaccinator of the island of Trinidad, contracted smallpox six months after a very severe revaccination.

(1) *This and other information is set forth in the remarkable book: Béchamp on Pasteur by Mrs. Douglas HUME. 3rd ed. C. W. Daniel Company, Ashington, Rocheford (Essex).*

This is formal proof that Jenner's vaccination does not protect against smallpox and that it even greatly increases mortality.

The smallpox and vaccinia viruses are not invisible as is commonly believed. They are both a mold whose 6 to 12-micron spores and mycelium abound in the pustules. The mycelium, very aggressive, causes a permanent mycosis and, by vegetating in the organs, causes various disorders, mental illnesses, illnesses of the nervous system, blood vessels, liver, kidneys, etc.

The vaccination practiced in France in 1947 was often severe and followed by accidents, particularly in Savoy where the death of several children forced vaccinations to stop.

As the Jennerian vaccine does not protect against smallpox, is the cause of accidents, and always causes a dangerous permanent mycosis, it must be abandoned and, above all, the obligation to vaccinate must be removed, as happened in England and Holland.

SUMMARY

The facts just presented prove to the reader that vaccinations are nothing more than a long deception cleverly organized to achieve the prosperity of a commercial business. The vaccine trade began in 1881 with the sale, by Pasteur, of the anthrax vaccine discovered in 1880 by Toussaint.

In 1885, Pasteur's total medical and biological incompetence was manifested by the mystification of the supposed cure of young Meister and rabies by an absurd, useless, and criminal treatment that senseless, charlatan publicity presented to universal admiration as the most brilliant discovery of modern times, but which, in three months (in 1886), killed 11 people by paralytic rabies inoculated by the vaccine.

In 1886, Pasteur created a limited company for the treatment of rabies, with a capital of 27,000 francs. A few years later, it marketed the anti-tetanus serum, which was a second mystification and a gigantic deception, since it was guaranteed a preventive action that the disease itself does not possess because it does not immunize against a second attack, a fact known at the time and which implied the impossibility of a preventive action of a serum.

In 1923, diphtheria vaccination began, which Zæller's experiment proved catastrophic in 1925, but whose results were knowingly distorted in order to continue selling the vaccine despite its harmful effects.

Then, in 1927, it was the Academy of Medicine's inaccurate assertion of the safety of the diphtheria vaccine and its desire to generalize its use that was made mandatory for the army.

And finally, from 1935 to 1938, it was the assertions, rigorously contrary to the facts, notably that this catastrophic vaccination would eliminate diphtheria in France, that distorted the opinion of the members of Parliament and made them vote for the odious law of June 25, 1938, which caused the current dreadful catastrophe: approximately 150,000 diphtheria inoculations and more than 15,000 children murdered since 1941, scathing and bloody proof of the falsity of the assertions made in Parliament and of the safety of the vaccine attested by the Academy of Medicine.

This sinister picture is completed by the harmful effect of the BCG vaccination, which is an error as considerable as that of the rabies vaccine and the serum and tetanus vaccine, an error due to ignorance of the microscopic anatomy of tissues and the application of false principles.

In summary, it is through a series of disasters for the public that the harmful effect of successive vaccinations has manifested itself.

Faced with such enormous, absurd, criminal errors, faced with the dire situation they have created, will no one intervene to protect the defenseless public?

Are we going to continue to inoculate paralytic rabies into all those who undergo the criminal anti-rabies treatment?

Are we going to continue the gigantic deception of virulent and ineffective anti-tetanus serums and vaccines against a disease that itself does not immunize?

Are we going to continue the BCG inoculation, which, being virulent and ineffective, is one of the biggest medical errors?

A young woman who murdered her child has just been sentenced to death, but more than 15,000 children have been murdered with impunity since 1941 by those who, in 1925, knowingly falsified the results of Zæller's test. By denouncing the catastrophic effects of diphtheria toxoid, Zæller imperiously ordered its definitive abandonment. Will the Government, Parliament, and the Judiciary allow this monstrous crime to continue?

We saw on page 9 that the High Council of Hygiene has just prepared a nice little law to make typhoid vaccination compulsory, a law that would realize the golden dream of bringing more than a billion in profits into the vaccine trade's coffers (30 million subjects at a gain of 50 francs each). The absurdity of this dangerous project was demonstrated on page 9.

Put into practice, it would cause a catastrophe as criminal as that of diphtheria toxoid.

A similar golden dream was already born last year with a wish from the Academy of Medicine to generalize tetanus vaccination, a dream that I shattered with an open letter informing the members of this Academy that this vaccination is absurd and, along with

the antitetanus serum, a gigantic and global deception that has dishonored French science for fifty years since the tetanus disease itself does not immunize.

If such a golden dream has already succeeded in coming true once, it is thanks to the culpable distortion of Zæller's control and the false claims that odiously deceived members of Parliament in 1936 and 1938. The enormous profits this criminal operation brought in have cost the nation since 1947 more than 150,000 children with serious diphtheria inoculations and more than 15,000 murders. Let the Members of Parliament remember this when they have their ballot papers in hand.

To believe that the French will accept this new attack on their bodily freedom and its serious risks denotes a state of unconsciousness and madness dangerous for the public, which will demand, on the contrary, to recover the freedom stolen from it in 1938 by odiously deceiving Parliament. And it will recover it.

I repeat that the law of June 25, 1938, has become obsolete because the current catastrophe is contrary to the intention of the legislator and places the French in a state of self-defense against vaccinations and in the right to refuse them, the law requiring them to receive a harmless vaccine that protects against the disease, but not a vaccine that causes it and kills.

You have only this means, and that of forming a powerful league to defend your children and yourselves. Among the French, will there not be some who want to take the initiative of a movement to found, in each department or region, a section of a national league for protection against deadly vaccinations and, for France, a central committee with a general secretariat and a publicity body whose means of action will be ensured by a small membership fee.

May all parents of children murdered, paralyzed, or made ill by diphtheria toxoid, may all parents of victims of anti-rabies, anti-tetanus, BCG, smallpox, typhoid, etc. serums and vaccinations, who number at least 250,000 to 300,000, may all those who have children threatened by vaccinations, rise up and unite to defend themselves by forming a national league with the help of the departmental or regional press. The dangerous typhoid and BCG vaccines are waiting for you. Act.

NOTICE

Professor J. Tissot informs that he does not have the material means to cope with the enormous correspondence that this brochure could generate. He advises his readers to first form groups of several hundred people who are members of the league and then to request the desired information by letter (with a stamp for the reply). They will request free insertion in one or more daily newspapers in the region, which will certainly not refuse it for a question of such vital importance to the public.

* * *

''I think if there are two men on Earth
that should have never been born,
it was Louis Pasteur and me.''

*Quote from the book ''*The Man W.H.O. Created AIDS*''*
by LANOO (Christian Anders) – 1993

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